A DESCRIPTIVE STUDY OF CHRONIC LEG AND FOOT ULCERS

Dissertation submitted to

THE TAMILNADU
DR. M.G.R MEDICAL UNIVERSITY, CHENNAI

With Partial fulfillment of the regulations

For the award of the Degree of

M.S (General Surgery)
Branch - I

GOVERNMENT KILPAUK MEDICAL COLLEGE
CHENNAI

MARCH 2010

CERTIFICATE

This is to certify that this dissertation is the bonafide work of **Dr. VADIVELU. P** on **"A DESCRIPTIVE STUDY OF CHRONIC LEG AND FOOT ULCERS"** during his course in M.S. General surgery from May 2007 to March 2010 at Government Kilpauk Medical College and Government Royapettah Hospital, Chennai.

Prof. Dr. S. UDAYAKUMAR M.S.
Professor and Head of the department,
Department of General surgery,
Govt. Royapettah hospital,
& Kilpauk medical college
Chennai -10

Prof. Dr. R. KUMAR M.S.
Professor of surgery,
Govt Royapettah hospital,
& Kilpauk Medical College,
Chennai -10

Prof. Dr. V. KANAGASABAI, M.D.,
DEAN,
Kilpauk Medical College, Chennai - 10.

ACKNOWLEDGEMENTS

I am most pleased to acknowledge **Prof. V. KANAGASABAI, M.D.**, Dean, Kilpauk Medical College and Hospital for the opportunity to conduct this study in the Department of surgery, Government Royapettah Hospital, Kilpauk Medical College, Chennai.

My deepest gratitude to my guide and mentor, **Prof. R. KUMAR. M.S**., Chief of surgical unit III, who has inspired me immeasurably during my training as a postgraduate student.

I also acknowledge the invaluable advice and counseling received from **Prof. S. JEYAKUMAR, M.S.,** Department of surgery, Govt. Royapettah Hospital.

I am very grateful to **Prof. S.UDAYAKUMAR M.S.** Head of the Department of General surgery for the encouragement and unrestricted permission to use the department of surgery.

This study would have not been possible without the support of our assistant professors, **Dr. A.K.RAJENDRAN, D.Ortho. M.S.,** and **Dr. P.MADHIVADHANAM, D.L.O, M.S.,** to whom I owe my surgical training.

I wish to express my gratitude to my **Co Post-Graduates** and my colleagues for their invaluable help in collection of patient data.

Not the least, I thank all **My Patients** around whom all our work revolve and towards whom our entire efforts trend.

A DESCRIPTIVE STUDY OF CHRONIC LEG AND FOOT ULCERS

CONTENTS

INTRODUCTION

INTRODUCTION

Chronic ulceration of the lower leg and foot is frequent condition and wide in distribution they may be associated with a number of Medical, Surgical & Dermatological condition the patient suffering is very immense, commonly seen in most of the surgical wards and OPD. The incident of ulceration is more in aging population and increased risk factor for atherosclerotic occlusion such as smoking, obesity and Diabetics. Ulceration can be defined as wounds with "full thickness depth" and a "slow healing tendency" in general the slow healing tendency is not simply explained by depth and size.

The problems of leg ulcer represent a wide spectrum of etiology, pathology, severity and morbidity. The main causes are venous insufficiency, lower extremity arterial disease and diabetes. Less frequent conditions are infections, vasculities, skin malignancies and ulcerating skin diseases such as pyoderma gangrenosum. But even rare condition exists such as recently discovered combination of vasculities and hypercoagulability. For a proper treatment of patients with leg ulcers, it is important to be aware of the large differential diagnosis of leg ulceration.

During the past three decades considerable knowledge has been gained regarding the physiology, anatomy, pathology and management of

chronic leg ulcers. Despite all this the management of chronic leg ulcers is a fertile field for experimentation. It is common to see patients with different types of ulcers due to various etiology and underlying systemic diseases. More over, leg and foot ulcers form a good bulk of patients in our hospital. Treatment of these ulcers forms a challenging task as well. I have therefore in my present study attempted to analyze the ulcers of the leg and foot.

This study comprises of review of literature with regard to historical aspects, etiology, anatomy, pathology, pathophysiology, clinical features and diagnosis of chronic leg ulcers along with the various modern investigative studies required for the diagnosis. Clinical investigation and histopathological study of patients of chronic leg ulcers admitted in Govt. Royapettah Hospital, Kilpauk medical college, Chennai. During the study period between June - 2007 to Nov - 2009.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

- To compare and analyze the distribution of age, sex, systemic disease and location of the leg and foot ulcers among 200 cases of the study group.
- To study the clinical features of various types of leg and foot ulcers.
- To study the usefulness of applied investigations.
- To effectively manage the condition.
- To prevent possible leg ulceration in high-risk individuals prone to the condition.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

I. HISTORICAL ASPECT OF LEG AND FOOT ULCERS

The incidence of chronic leg ulcer is as old as history, as with any disease of mankind. The most common and noted ulceration of the chronic leg ulcers for many years is Stasis ulcers. Hippocrates, the legendary father of medicine himself had a leg ulcer. He treated multiple varicose veins by puncturing them at different levels to avoid non-healing of ulcers and about 400 years B.C. He wrote - " In case of an ulcer, it is not expedient to stand, especially if the ulcer be situated on the leg" (Sarkar P. K. Ballantynes).[1]

It was John Gay (1968) who first noticed that varicose veins and leg ulcers were often associated.In 1828 Marjolin described the carcinomatous ulcers originating from degenerative burns scars - this ulcers bears his name. Avicenna (982 - 1027 AD) gave a good account of diabetes and was the first one to describe diabetic gangrene.In 1909 Burger described a syndrome of vascular occlusion in which arteries, veins, and nerves of the extremities were involved in extensive fibrosis that resulted in ulceration.

Although a minor traumatic incident is often the immediate cause of the ulcer, the underlying pathology is usually vascular. Callam et al (1985) estimated that 70 percent of leg ulcers are venous, 10 per cent are arterial and 10-15 per cent are of mixed arterial/venous origin. Other causes include malignancy, neuropathy, pyoderma gangerenosum and sickle cell anaemia.

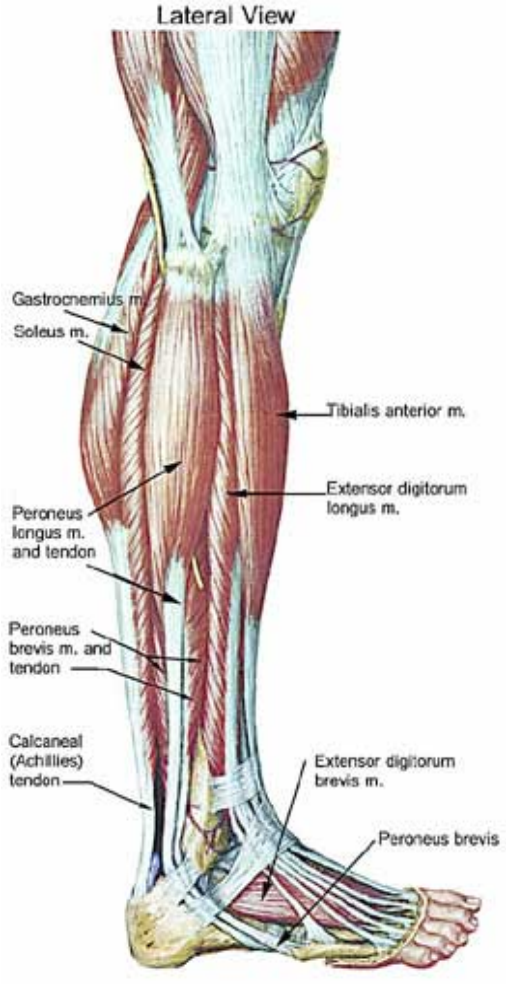
II. INCIDENCE AND OCCURRENCE OF LEG AND FOOT ULCERS

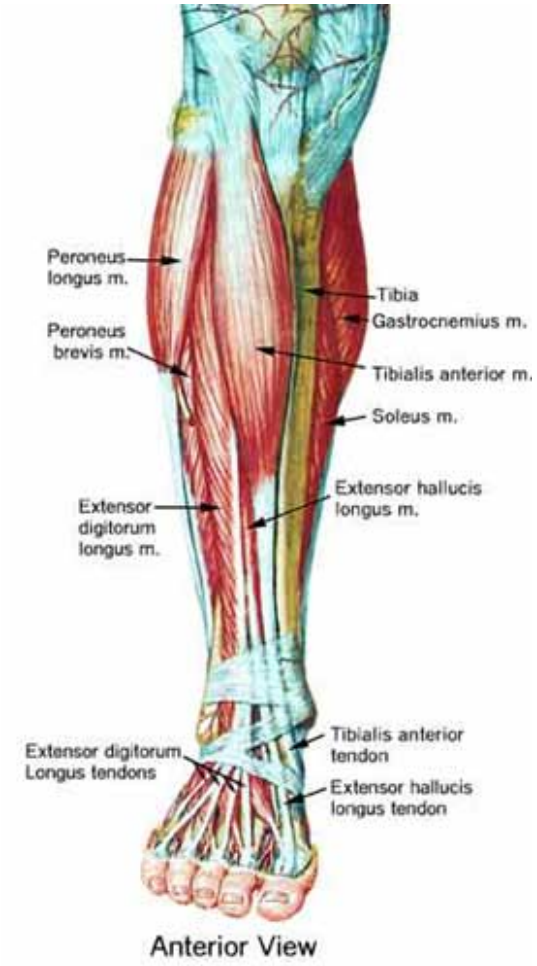
Accurate data concerning the incidence of non-fatal diseases are difficult to obtain and statistics are usually derived from hospital attendance records and general practice surveys. The prevalence of a condition within a community may be affected by many factors such as the genetic constitution of its members, nutritional status, environmental factors, customs and occupation.

The prevalence of leg ulcers is probably between 0.18% and 1% of the population. (Phillips Tania et al).**2** The site of ulceration is recorded using the method of Callum; 90% of the ulcers were present in the gaiter area, 2 % in the foot and 8% in the leg (Baker S. R. et al).**3** Incidence of ulcers is more common among women with no difference in age related prevalence.

A basic study of 4422 healthy working adults aged between 20 and 70 years in Europe resulted in detection of chronic venous insufficiency in 19% of men and 25% of women. Information from other parts of the world other than Europe and America is sparse, but it is generally held that ulcerations due to venous insufficiency are less common in African natives. Park (1969) compared the incidence of varicose eczema in their middle aged Indian patients (7%) with that in comparable "white" patients (3%) and Bantu (1%) but give no figures for the incidence of venous ulcerations.

ANATOMY OF LEG





III. ANATOMY OF THE LEG AND FOOT

The leg is that part of the lower limb below the knee and the terminal part of the leg below the ankle is the foot.

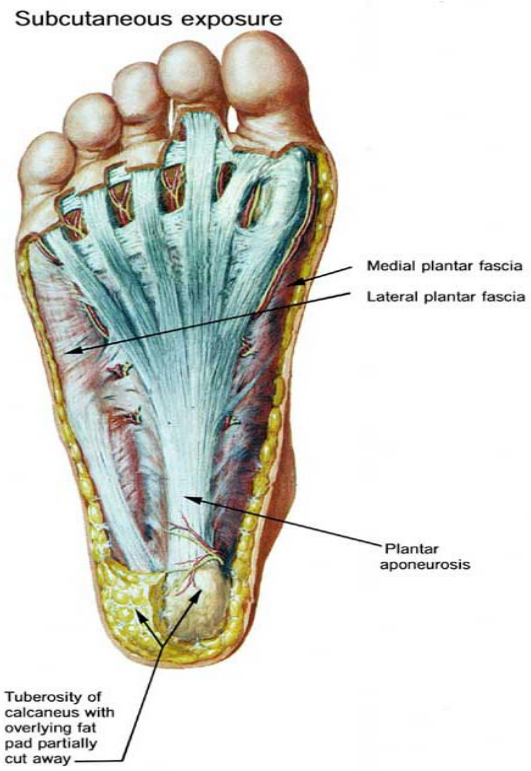
Front of the leg and dorsum of foot

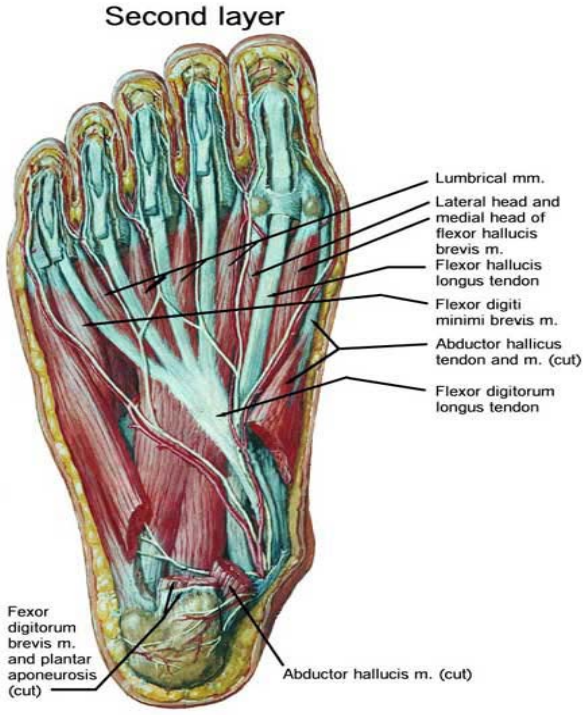
Superficial fascia of the front of the leg and the dorsum of the foot contains the following parts:

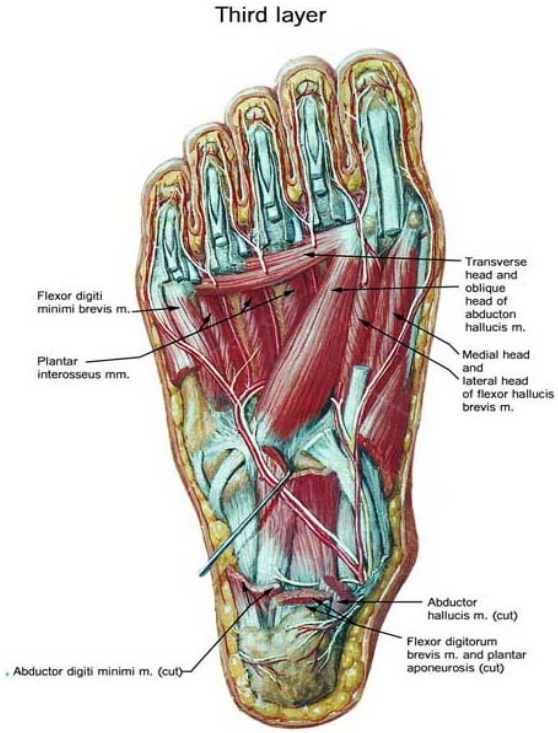
- ❖ Superficial veins.
- ❖ Cutaneous Nerves
- ❖ Lymphatics
- ❖ Small unnamed arteries.

The subcutaneous bony surfaces are not covered by the deep fascia of the leg but are attached to it at its borders. It is thick in the upper part of the leg and gives origin to the underlying muscles while in the lower part is thin and forms retinacula around the ankle. Intermuscular septa are formed in the deep fascia, which tends to divide the leg into compartments. The anterior and the posterior intermuscular septa are attached anterior and posterior borders of the fibula and divide the leg into anterior, lateral and posterior compartments. In the posterior compartment a superficial transverse facial septum separate tibialis posterior from the long flexors of the toe.

ANATOMY OF FOOT







Anterior compartment of the leg

Muscles are Tibialis anterior, Extensor halucis longus, Extensor digitorum longus and Peroneous tertius.

Anterior Tibial Artery

The main artery that is found in the anterior compartment of the leg is the anterior tibial artery. The perforating branch of the peroneal artery feeds this artery. Anterior tibial artery is the smaller terminal branch of popliteal artery.

Branches

a) The adjacent muscles are supplied by the muscular branches.

b) Anastomotic branches feed the knee and the ankle. Anterior and posterior tibial recurrent branches take part in the anastomosis around the knee joint. Anterior medial malleolar and anterior lateral malleolar branches take part in the anastomosis around the ankle joint.

Dorsalis Pedis Artery

The direct continuum of the anterior tibial artery, which forms the chief artery of the dorsum of the foot, is the dorsalis pedis artery.

Branches

1. Lateral tarsal artery supplies extensor digitorum brevis, tarsal joints and ends in lateral malleolar network.

2. Medial tarsal branches joints medial malleolar network.
3. Arcuate artery is a large branch that arises opposite the medial cuneiform bone. It gives off second, third and fourth dorsal metatarsal artery each of which divides into dorsal digital branches for the adjoining toes.
4. First dorsal metatarsal artery gives a branch to medial side of big toe and divides digital branches for adjacent sides of first and second toes.

Cutaneous Innervation

1. Saphenous nerve
2. Lateral cutaneous nerve of calf
3. Superficial peroneal nerve
4. Sural nerve
5. Deep peroneal nerve
6. Digital branches of the medial and lateral plantar nerve

Deep Peroneal Nerve

This is the nerve of the anterior compartment of the leg and the dorsum of the foot. It is one of the two terminal branches of peroneal nerve.

Branches

1. The adjacent sides of the first and the second toes are supplied by the cutaneous branches.

2. The muscles of the anterior compartment of the leg and the extensor digitorum brevis on the dorsum of the foot are supplied to by the muscular branches.
3. Articular branches supply the ankle joint, tarsal joint, tarsometatarsal and metatarsophalangeal joint of the big toes.

Lateral Side of the Leg

The lateral compartment of the leg is a bound unit. Anteriorly by anterior intramuscular septum, posteriorly by posterior intramuscular septum medially by lateral surface of fibula.laterally by deep fascia of the leg.

• Muscles of this compartment are Peroneus longus and Peroneus brevis.

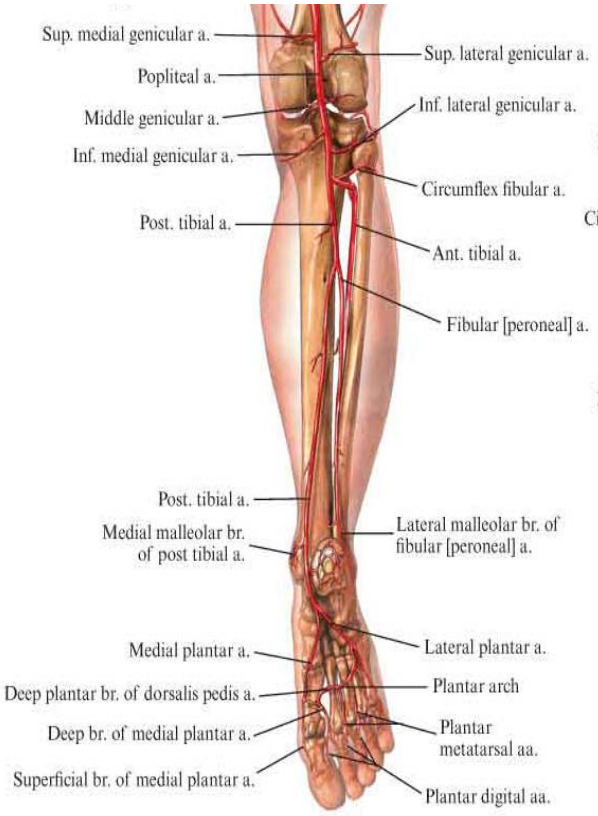
• Arterial supply is derived from the branches of peroneal artery; veins drain into small saphenous vein.

• Nerve supply by Superficial peroneal nerve, branch of common peroneal nerve.

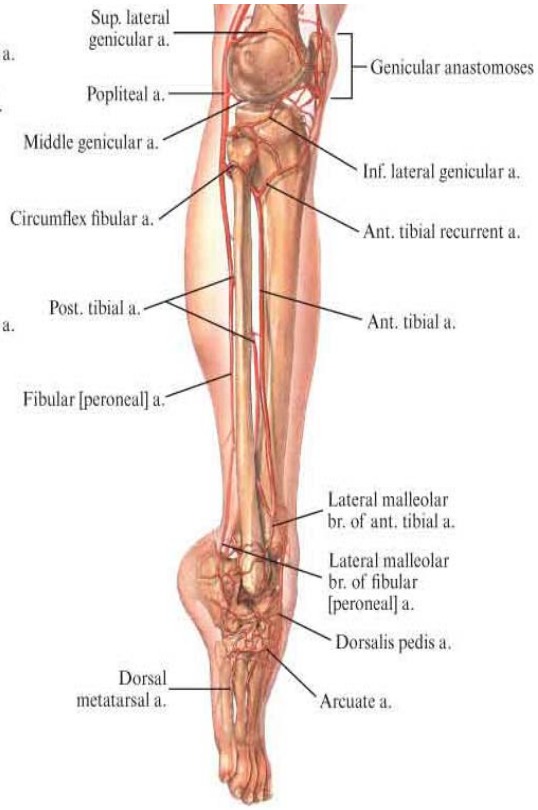
Medial side of the leg

Medial surface of the leg is covered only by the skin and the superficial fascia and hence this part is largely subcutaneous. The great saphenous vein lies in the superficial fascia as they cross the lower 1/3 of this surface. This skin, fascia and the periosteum of this surface are supplied by saphenous nerve.

ARTERIAL SUPPLY OF LEG AND FOOT

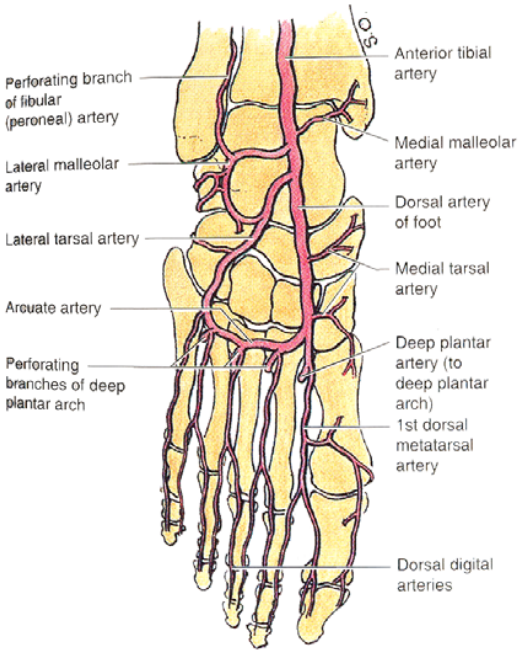


Posterior View

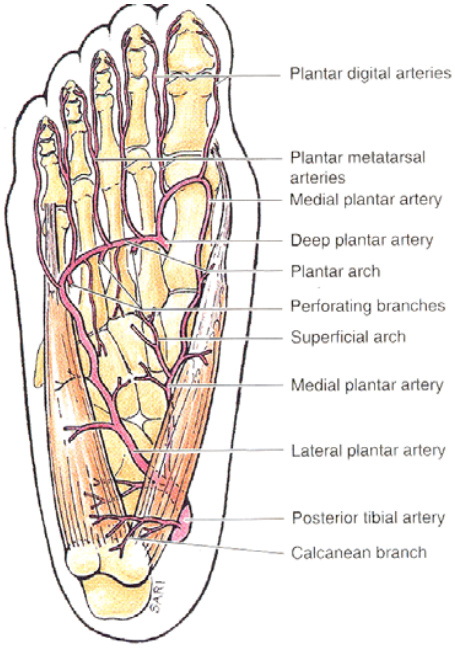


Lateral View

ARTERIES OF THE FOOT



(A) Dorsum of foot



(B) Plantar aspect of foot

The Posterior compartment of the leg.

Superficial fascia of the back of the leg contains the small and the great saphenous vein, several cutaneous nerves, medial and lateral calcaneal arteries.

The muscles of the posterior compartment are divided into superficial and deep Group. The superficial muscles are the gastrocnemius, Soleus and the Plantaris. The deep muscles are the Popliteus, Flexor digitorum longus, Flexor hallucis longus and the Tibialis posterior.

- **Posterior Tibial Artery**

This is the larger terminal branch of the popliteal artery.

Branch

- Peroneal artery - the largest branch of the posterior tibial artery, supplies the lateral and the posterior compartments of the leg.
- Muscular branches for the posterior compartment.
- Nutrient artery to tibia is the largest nutrient artery in the body.
- Anastomotic branches – Circumflex fibular for the knee, communicating branch for peroneal, medial malleolar and calcaneal artery for ankle and heel.
- Terminal branches – medial and lateral plantar.

Cutaneous Innervation

1. Saphenous nerve
2. Posterior division of the medial cutaneous nerve of thigh
3. Posterior cutaneous nerve of the thigh
4. Sural nerve
5. Lateral cutaneous nerve of calf

Sole of the foot

The skin of the sole of the foot is thick, firmly adherent to the underlying plantar aponeurosis and is creased.

Cutaneous innervations

A. Medial calcaneal branches of tibial nerve
B. Branches from medial plantar nerve
C. Branches from lateral plantar nerve

Superficial fascia

This is more fibrous and dense. The fibrous brands bind the skin to the deep fascia and divide the subcutaneous fat into small tight compartments. The fascia is very thick and dense over the weight bearing points.

Deep Fascia

Deep fascia of the sole forms:

a) Plantar aponeurosis in the sole.

b) Deep transverse metatarsal ligaments between the metatarsophalangeal joints.
c) Fibrous flexor sheaths in the toes.

Muscles of the first layer of the sole are Flexor digitorum brevis, Abductor hallucis and Abductor digitorum. The muscles of second layer are Flexor digitorum accessories and lumbricals, which are four in number. Muscles of third layer of sole are Flexor hallucis brevis, Adductor hallucis. and Flexor digiti minimum brevis. The fourth layer of the sole contains the muscles – the Dorsal interossei which are four in number and lie between metatarsal bones and the Plantar interossei which are three in number and lie below the metatarsal bones.

The main arteries of the sole are the medial plantar artery and the lateral plantar artery.

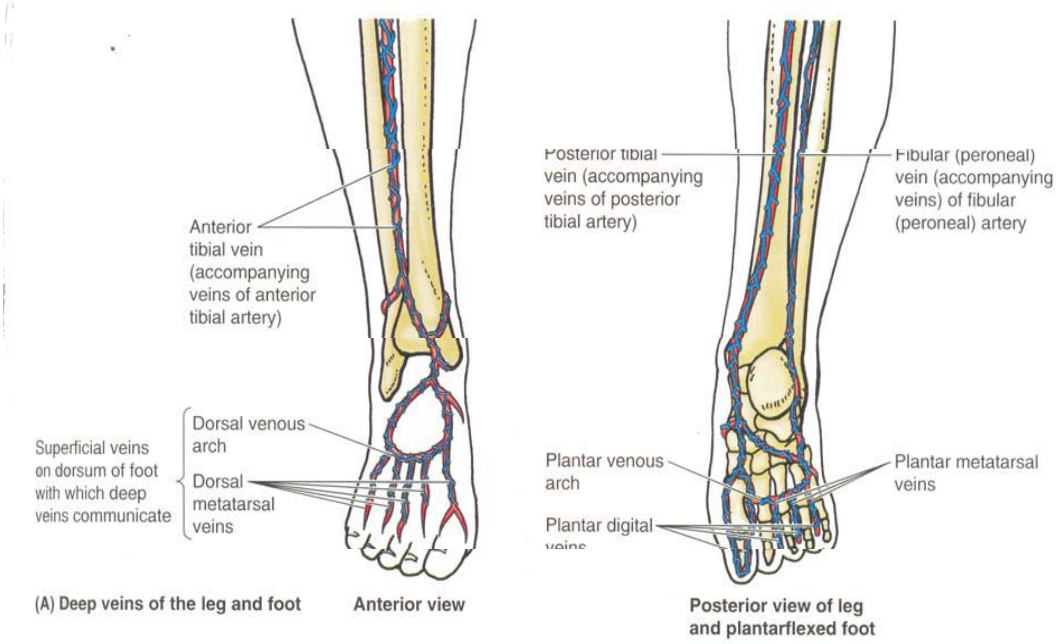
The main nerves of the sole are medial and lateral plantar nerves.

Venous drainage of the leg and foot

Superficial veins

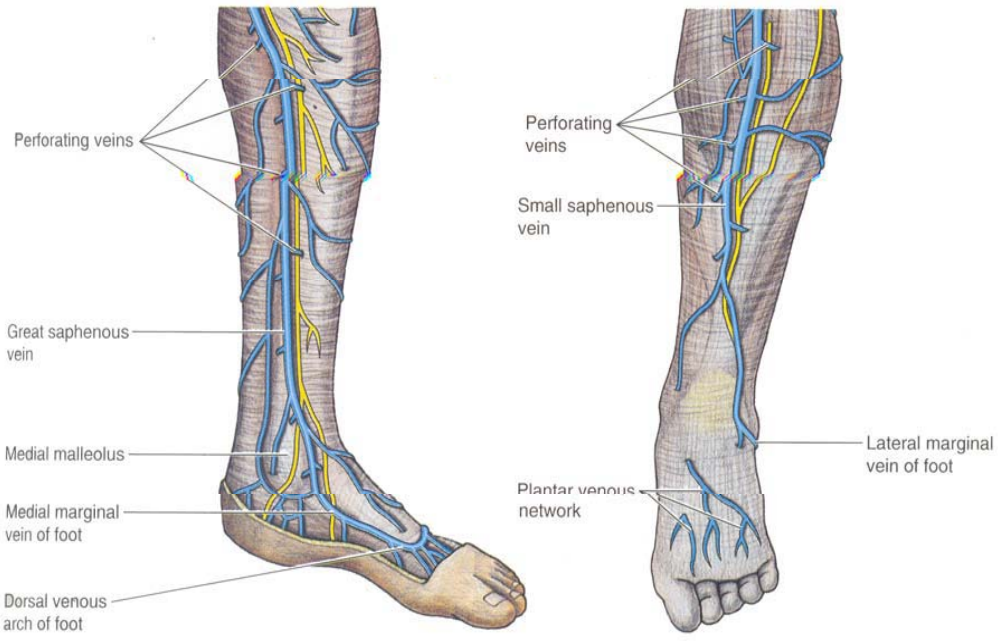
These are the long and short saphenous systems and their tributaries. They lie in the superficial fascia.. A large portion of blood from the superficial veins is drained into the deep veins through perforators.

SUPERFICIAL AND DEEP VEINS OF THE LEG AND FOOT



(A) Deep veins of the leg and foot — Anterior view

Posterior view of leg and plantarflexed foot



(B) Superficial veins of the leg and foot — Medial view

Posterior view of leg and plantarflexed foot

The Dorsal venous arch

This is formed by the four dorsal metatarsal veins, each of which is formed by two digital veins

The long saphenous vein

This is formed by the union of medial end of dorsal veins are which medial marginal vein, from the medial side of the great toe and drains into the femoral vein.

The short saphenous vein

This is formed by the union of the lateral end of the dorsal venous arch with lateral marginal vein from the lateral side of the little toe and opens into popliteal vein.

The deep veins

a) The anterior tibial.

b) The posterior tibial.

c) The peroneal.

d) The popliteal.

These veins accompany their corresponding arteries. They are supported by the surrounding powerful muscles.

Perforating Veins

These are of two types:

a) The direct perforators – these connect the superficial veins to the deep veins. The long and short saphenous veins are the large direct perforators
b) The indirect perforators – these connect the superficial veins to the deep veins through muscular veins.

LYMPHATIC DRAINAGE

Superficial lymphatics

These form large trunks and are more numerous than the deep lymphatics. They run in the superficial fascia in two streams.

a) The main stream follows the long saphenous vein and ends in the lower vertical group of the superficial inguinal lymphnodes.
b) The accessory stream follows the short saphenous vein and ends in the popliteal lymph nodes.

Deep Lymphatics

These are fewer than superficial lymphatics and drain all structures lying deep tothe deep fascia. They terminate mostly into the deep inguinal nodes either directly or indirectly through popliteal nodes.

IV. BIOLOGICAL PROCESS OF HEALING

In the wound healing process three distinct phases can be visualized.

1. The phase of inflammation (Day 1-4)

2. The proliferation phase (Day 5-20)

3. The differentiation phase (Day 20 onwards).

The inflammation phase

Contact of the collagen with the blood flowing in the wound causes the activation of kinin and complement cascade, which is the initial phase in the clotting process. The blood vessels undergo brief constriction followed by vasodilatation under the influence of histamines from the platelets and mast cells. An increase in the capillary permeability is noted. White and red blood cells escaping from the walls of the blood vessels form a network of fibrins over the wound site, which within three hours is surrounded by a few lymphocytes and an increasing number of lytic polymorphs. By the fifth day monocytes become the dominant cell type. By the end of the first phase new capillaries bud from the endothelial cells in the capillaries near the wound edge. Clinically in this phase the classical features observed that are manifested by the wound are heat, redness, tenderness, swelling and loss of function.

The Proliferation phase

By the fifth day fibroblasts have began to synthesize collagen and Ground substance.

The differentiation phase

During this phase, rationalization of copious new blood vessels and notably a remodeling of the haphazard arrangement of the collagen fibers take place.

Healing by first intention

If wound edges are approximated, healing proceeds rapidly to closure. This is called healing by first intention.

Healing by second intention

When the wound edges do not come together or when there is irreparable skin loss or when the wound becomes infected, then in such cases healing takes place by second intention. In this type of healing, the wound is healed by a filling of granulation tissue.

Other methods of providing skin cover – in the presence of devitalized tissue, swelling tension and skin loss – include delayed primary suture, skin grafting and second suture

Factors contributing to rate of wound healing

Blood Supply: Good blood supply is necessary for early healing.

Tension: Tension of the tissue inhibits the blood supply resulting in wound failure.

Age: Ageing causes decreased protein turnover leading to slower wound Healing

Mobility: Delicate blood supply is easily damaged by movement and shearing forces.

Infection: Infections are a major factor in wound healing failure.

Malnutrition: Defective collagen and ground substance synthesis can be directly linked with malnutrition. Severe protein calorie malnutrition has long been implicated in the failure of wounds to heal.

Uremia: Clinically uremia is implicated in the retardation of wound healing.

Jaundice: Jaundice is associated with reduction in wound strength. Biopsies of the skin in jaundice patients show a reduction of the enzyme proline - hydroxylase involved in the collagen maturation.

Steroids: Steroids depress wound healing by their anti-inflammatory action.

Radiation: Radiation causes cell death by damaging both DNA and Disrupting intracellular metabolism.

V. AETIOLOGY AND PATHOLOGY

About 95% of the leg ulcers are due to vascular aetiology (Gilliland)[4] and venous ulcers account for up to 90% of cases (Burton. S. Claude)[5], (Callam MJ, et al)[6]. Arterial disease accounts for 5-10%; most of the others are due to neuropathy, usually diabetic or a combination of these diseases (Yound JR).[7] Diabetic ulcers are common on the toe and the heel (Hansson Carita).[8] In patients suffering from arteriosclerosis and hypertension, arterial ulcers are on the increase, especially in the western countries. Arterial insufficiency and / or diabetes may also be the causatives for ulcers below the line of the shoe. Ulcers at the ankles in the gaiter zone and venous ulcers are mostly caused by venous insufficiency (Hansson Carita).[8] Another important causative of venous ulcers is sapheno-femoral incompetence (94.4%) (Sethia. K and SG Drake).[9] Primary varicosity of the long saphenous system and / or short saphenous system is one of the causative factors for venous ulcerations (Hoare MC et al).[10]

The elevated ambulatory pressure in peripheral venous system in chronic venous insufficiency manifests itself not only in form of disturbed microcirculation but also and particularly in microangiopathic changes. These include decrease in capillaries, glomerulus like changes and decrease in oxygen content. (Junger, M Stiens. A).[11] It has been also noted that perivascular fibrin cuffs and skin hypoxia precede lipodermatosclerosis in

limbs at increased risk of developing a venous ulcer (Stacey M. C., Burnand K.G et al).[12]

CLASSIFICATION OF LEG AND FOOT ULCERS

a. Venous ulcerations
b. Arterial insufficiency – Thromboangiitis Obliterans, atherosclerosis, Raynaud's disease and phenomenon, hypertensive ischaemic ulcer (Martorell's ulcer) and embolic occlusions (sub acute bacterial endocarditis).
c. Diabetic neuropathic ulcerations.
d. Neoplastic ulcers – epithelioma melanoma, basal cell carcinoma and malignant change in long standing scars (Marjolin's ulcer).
e. Tropical ulcers including leishmaniasis, fungal infections.
f. Specific infections – tuberculosis, syphilis and AIDS.
g. Blood dyscrasias – severe anemia, sickle cell anemia, thalassemia, hereditary spherocytosis and leukaemia.
h. Nutritional and metabolic disturbances.
i. Skin sensitivity or allergy.
j. Trauma.
k. Necrosis by injection of chemicals, insect bites, snakebites or radiation.
l. Repeated trauma.

VENOUS ULCER

DIABETIC ULCER

TRAUMATIC ULCER

ARTERIAL ULCER

TROPHIC ULCER

MALIGNANT ULCER

Rheumatoid arthritis.

m. Systemic autoimmune and micro vascular diseases.[13]

The Venous Ulcer

Histopathologic reasons caused by venous insufficiency, is one of the main causes of venous ulceration. Changes taking place in the cluster of thick walled capillaries within a thickened fibrotic papillary dermis, accompanied by siderophages and extravasated erythrocytes. Occurrence of perivascular fibrin cuffs in the skin is seen in venous insufficient areas. In venous ulcers it should be noted that the fibrin and the neutrophils are present in the walls of the vessels in the granulation tissue beneath the ulcers. Lipodermatosclerosis or sclerosing panniculitis demonstrates fibrosis and thickening of the subcutaneous septa. An early lymphocytic infiltrate of the subcutaneous septa is gradually replaced by a mixed infiltrate with increasing fibroplasis and sclerosis.

Dysfunction of the calf muscle pump can result in venous insufficiency in the deep, connecting or superficial veins; arteriovenous fistulae, or muscle dysfunction as a result of fibrosis, neuropathy or inflammatory diseases. Valvular damage caused due to thrombosis may lead to deep venous insufficiency. Venous ulcers are mainly attributed to be

caused by venous insufficiency; however multisystem incompetence of the valves is also a common cause.

Increased venous pressure causes leakage of fibrinogen and white blood cells through widened endothelial pores. These cells release inflammatory mediators increasing vascular permeability. This causes tissue ischemia and ulceration.

Arterial Ulcers

Arterial or ischaemic ulcers are most commonly due to atherosclerosis and hence encountered in older adults. They can be also seen in younger adults and here usually peripheral arterial disease like Thromboangiitis Obliterans is the cause. The other rare causes of ischaemic ulcers are Raynaud's diseases and phenomenon.

Atherosclerosis develops at twice the frequency in patients who smoke compared with non-smokers (Coffman J. D.)[14] about 50% of the patients have lipoproteinemia. Patients with diabetes develop the disease at an earlier age than non-diabetics and have more severe and progressive disease.

There is focal accumulation of lipids, mucopolysaccharides, blood and blood products, fibrous tissue and calcium deposits in the intima of the arteries. The media becomes atrophic with thin strands of muscles, disrupted

elastic lamella, collagen tissue and calcium deposits. Enlarging plaques encroach upon the lumen despite dilatation of the artery, and plaques may ulcerate. Hemorrhages occur within the arterial wall. Thrombi are formed and finally occlude the vessel lumen.

Another important feature here which causes sudden gangrene with ulceration is atheromatous embolisation. It is due to embolisation of small pieces of atheromatous plaques and debris to the arteries of extremities. This is called as blue toe or trash foot syndrome.

Another important arterial disease, which can cause ulceration and gangrene, is Thromboangiitis Obliterans (Buerger's disease).

The etiology of TAO remains unknown. Almost all patients who develop this disease are smokers and the syndrome sometimes abates following cessation of smoking. An increased frequency of HLA-A9 and HLA-B8/B5 have been reported but not found by all investigators (Millis J. L. et al).[15] The pathogenesis involves production of ischaemia and all its manifestations by an inflammatory action of medium and small arteries of extremities and also obstruction by thrombi.

Diabetic Ulcers

The various factors, which are the contributions for the cause of ulcers, are Hyperglycemia, microangiopathy, Neuropathy, Liability to infection and Alteration in blood flow.The precipitating factors are injuries and infection.

In a diabetic condition of either type 1 or type 2, hyperglycemia may lead to surgical complications. In this condition it affects the basement membrane of the capillaries and cell permeability; interfering with the transfer of oxygen and nutrients to the tissues. Along with decreased supply of leukocytes into this area and also high concentration of glucose in the tissue fluids which help in the growth of pathogenic organisms, it impairs wound healing. The principle finding in the vascular flow mechanism in diabetes is a marked decrease in blood flow as shown by Doppler waveform. There is associated loss of the important diastolic back flow phase. These changes are also reflected in the high ankle / brachial Doppler ratio found in diabetes. The major cause of this blood flow change is the distal AV shunting and sympathetic dysfunction. The other cause being atherosclerotic stiffening of the vessels, sometimes this is associated with calcification of the vessel walls.

The thickening of the basement membrane is characteristic of both variants of diabetes mellitus. There is either increased synthesis of basement membrane or decreased degeneration. Diabetic patients also tend to develop calcifications more commonly and at a much younger age.

Histologically the disease is manifested as a thickening of the tunica intima along with large atheromatous plaques and lumps. The metatarsal arteries are more often occluded in diabetes. An important yet a very common factors responsible for producing neuropathic ulceration is the presence of diffuse distal peripheral polyneuropathy due to which painless ulcers are predominantly seen in the lower limbs. Osmotic and metabolic derangements are caused due to ischemia from vessel occlusion and altered capillary permeability; this paves the way for producing neuropathy. Increased capillary permeability allows the toxic proteins which are circulating to reach the nerves and tissue oedema which cause the impairment of nutrition contribution to development of the neuropathy.

Reduction or absence of sensation increases the changes of injury occurring to the foot from heat, sharp objects, pressure from ill-fitting shoes etc. In the most severely affected patients the chances of spreading infection, formation of large ulcers and gangrene is increased tremendously. The motor changes are primarily manifested in the small muscles of the foot.

Metatarsophalangeal joints are hyperextended when the small muscles of the feet are paralyzed with preserved function of the long flexors and extensors. Because of this condition there is a predisposition to the development of ulcers, especially at the tip of the toes from the abnormal pressure.

Trophic Ulcers

Trophic ulcers are particularly perforating ulcers of the foot, which is associated with Tabes dorsalis, diabetic neuropathy, leprosy, and other diseases of the central nervous system.

The three modalities of neurological deficit are:

1) Sensory loss.
2) Motor paralysis.
3) Autonomic nerve damage.

Due to autonomic neuropathy there is a failure of vasodilatation producing hypoxic environment predisposing to infection in minor injuries. The absence of the sweat glands or its malfunction, with the loss of Lysozymes alters the cutaneous bacterial environment and also decreases the defense mechanism of the skin. Trophic changes include redness and glossy shining skin with loss of hair. Thick keratinization of the skin and atrophy of the local subcutaneous tissue is observed in areas of increased pressure. Breakdown of such skin produces trophic ulcers.

Studies have shown that the initiation of the ulceration is usually due to an initial damage in the deep tissue surface. The pressure of the entire body falls on the foot and during walking, unperceived by sensation and the normal rotation of the joints is uncontrolled by muscular coordination, the result being the introduction of torsional stress, which leads to necrosis of the deep tissues immediately adjacent to the bone. The clinical features follow a sequence of incidents which are listed below:

1) Initial deep necrosis.
2) Necrotic sinuses.
3) Necrotic blisters and finally.
4) Plantar ulcer.

MALIGNANT ULCERS

Squamous cell carcinoma

This is the carcinoma of the cells of the epidermis that usually migrate outwards to the surface. The initiation point of the squamous cell carcinoma is usually the layer that is formed by the prickle cell layer. This may also occur in a few preexisting lesion of the skin like:

1. Long-standing chronic ulcers (e.g. Marjolin's ulcers) following burns, venous ulcers, old scars etc.
2. Senile keratosis.
3. Bowen's disease.

4. Leukoplakia.
5. From skin exposed to irradiation.
6. Chronic skin lesion e.g. Lupus Vulgaris (Cutaneous tuberculosis), eczema, warts.
7. Exposed to prolonged irritation by various chemicals.

Macroscopic Features

The origin of the squamous cell carcinoma is as a small lump or nodule. These nodules enlarge as they mature and become necrotic and their centers sloughs out. The edges of the ulcer are raised and everted; this shows excessive tissue growth just above the surface. The floor of the wound is covered by necrotic tumor, serum and blood. Presence of pale coloured unhealthy granulation tissue may also be seen. The base of the wound is usually indurated.

Microscopic Features

Solid columns of epithelial cells, which are seen, growing down into the dermis, separated from one another by connective tissue.. This mass of keratinized cells are surrounded by normal looking squamous cells presenting the characteristic ‘prickle cell’ appearance and these are arranged in collective manner as seen in ‘onion skin’. This whole arrangement is called as a ‘cell nest’ or ‘epithelial pearl’. This may be absent in rapidly growing tumor and in mucous membranes.

Malignant Melanoma

All the melanomas originate from the melanoblasts at the dermo-epidermal junction, but as the cells may not contain melanin at all times and therefore some lesion may be amelanotic.

The malignant melanoma may rise from a pre-existing pigmented naevus (90%) – either a junctional naevus, compound naevus or in Hutchinson's Lentigo.

Macroscopic Features

The epithelium lying just above the mole ulcerates and often breaks down with minor trauma, which makes the wound to bleed. Palms and the soles are the most common areas where melanomata are seen. The coloration may range from black to brown. If the wound is more blackish in colour the chances of it being malignant is more. When they are small, the surface is smooth but as it attains larges size, ischemic necrosis occurs and small ulcers and crusts etc, are seen on the surface. The surface of the big melanoma appears wet, soft and boggy yet the tumour feels firm.

Microscopic Features

Development of the malignant melanoma brings forth an increase in the functional activity and the cells increase in size. The nucleolus is enlarged, hyperchromatism is present and mitosis is observed. The

cytoplasm is often vacuolated with fine melanin granules to mimic paget cell. The invasion of the dermis is determined by the presence of polyhedral or circular cells with abundant spongiocytoplasm and fine pigment granules. The tumor cells may form clusters in the sub epidermal lymphatics. Invasion of lymphocytes which are inflamed are found in the sub epidermal zone. This is a very good indicator of a malignant melanoma. In fully developed melanoma the large tumor cells in the dermis often show alveolar arrangement the groups being separated by a fine stroma.

VI. CLINICAL FEATURES AND DIFFERENTIAL DIAGNOSIS

Chronic leg ulcers can be diagnosed based on the patient's medical history, appearance and location of the ulcer along with clinical examination and investigations. History is very useful in diagnosing ulcers caused by associated diseases. The diseases that have to be specially inquired for are Diabetes Mellitus, Syphilis, Tuberculosis, Nephritis, Liver disorder and Blood disease. The primary lesion might have been altered either by the use of ointments or by infections. Thus a history of eczema helps for diagnosis of stasis ulcers. History of trauma will help for evidence of underlying fractures or osteomyelitis. A nodular ulcer appearing after a thorn prick points to deep mycosis. A painful ulcer with undermined edge or ulcer healing at one end and growing at other end might be of tubercular origin. Ulcers growing on already existing moles may be malignant melanoma. Rolled out borders of chronic ulcers may be carcinomatous ulcer. Ulcers growing on scar tissue may be Marjolin's ulcers. Punched out ulcers without any symptoms in elderly people may be due to gumma. Knowledge of precipitating factors is also equally important. Ulcers that get aggravated during cold season indicate Raynaud's disease. Thus a clear idea of primary lesion of an ulcer and its later development helps one in diagnosis.

An understanding about the location of the leg ulcer is crucial. The area around the medical malleolus is drained by long saphenous venous system and communicating vein of lower limb and this is the area where occurrence of stasis ulcer is invariably seen. Hypertensive ulcers or ischaemic ulcers are usually pale with an eschar and are usually seen on the lateral side. Hyperpigmentation and or eczematization point towards stasis element. In Werner's disease or scleroderma, sclerotic skin formation is observed.

Atrophic skin formation around an ulcer indicates to acrodermatitis chronic atrophicans or an Atrophic Blanche. Occurrence of the gummatous ulcers is over the sub-cutaneous bones such as tibia, sternum etc. Distinguishing between a spreading ulcer and a healing ulcer is extremely important. Inflamed and oedematous edges of the ulcers indicate spreading ulcers. In a healing ulcer, the edge is traced from red gramulation tissue and at the center it will show a blue and a white zone. An undermined edge is usually observed in a tuberculous ulcer and punched out edge in gummatous ulcer. A pearly white beaded edge which is raised is characteristic of a rodent ulcer. An everted edge points to squamous cell carcinoma.

An understanding of the floor of the ulcer or exposed part of the ulcer is extremely important. Red granulation tissue covering the floor of the ulcer points to a healing ulcer. Smooth granulation with a pale coloration indicates

to a slow healing ulcer. Trophic ulcer penetrates down to the bone which becomes the floor in this case. Black mass on the floor suggests malignant melanoma.

Discharge

The quantity and the coloration of the discharge from an ulcer help in identifying the nature of the ulcer. Profuse discharge from an ulcer indicates to a healing ulcer whereas a purulent discharge points to a spreading ulcer. A greenish coloration to the discharge indicates to an infected ulcer and this may be caused by pyogenic bacteria. Serosanguinous or blood mixed discharge is pathognomonic of malignant ulcer, or else tuberculous ulcer.

Description of various ulcers

Arterial ulcers are rare compared to the venous ulcers. An arterially compromised limb can be pale and cyanotic. The skin is often dry and scaly or shiny and atrophic with brittle nails or loss of hair growth. The appearance of the ischemic erythema is usually on the foot. Atherosclerotic arterial ulcers are often seen in older people and when they occur in the young adults, it usually points to Thromboangiitis Obliterans and other forms of vasculitis. Pressurized areas of the foot are the focal points for the appearance of these ulcers. Toes, bony prominences or in pretibial and lateral malleolar areas are some of the common placer where these ulcers can be spotted. These ulcers have sharp defined edges and are punched out.

The ulcer destroys the deep fascia and may even expose the tendons at the base. The ischemic ulcers are extremely painful and the pain is excruciating irrespective of the size of the ulcer. The development of the arterial ulcers is more prolonged when compared to the venous ulcers. The appearance of ulcers having punched out edges with slough deployment on the floor of the wound resembling a gummatous ulcer points directly to a trophic ulcer. The most common forms of trophic ulcers seen are the bedsores. The heel and the ball of the foot and the back of the heel are the most common places where these types of ulcers occur. These ulcers start with callosity under which suppuration takes place, the pus comes out and the hole at the center forms the ulcer which gradually burrows through the muscles and the tendons to the bone. Tuberculous ulcer develop when cold abscess from bone and joint tuberculosis break out of the surface. Appearance of the ulcer is in the form of a thin edged one wound, which is reddish blue in colour and undermined. The base of the wound is coloured pale and there is slight induration at the base.

The areas where gummatous ulcer appear most commonly is in the upper part of the leg i.e., over the subcutaneous bones of the tibia. The wound has punched out edges and a yellowish grey gummatous tissue (wash lather slough) on the floor.

Erythrocyanoid ulcer is associated with ‘erythrocyanosis frigida’, which is found to occur exclusively in young aged women. Abnormally fat subcutaneous fat with thick ankles combined with abnormally poor arterial supply are the contributing factor for the formation of this type of ulcer. The skin of the ankle tends to become abnormally sensitive to temperature in such patients. When the temperature drops the ankle becomes cold and blue and often tenderizes. In hot weather chronic reactive hyperemia is evident and the ankle becomes hot, oedematous, swollen and painful. The patient is much more troubled by chilblains. Palpation of the leg reveals the presence of small, superficial and painful nodules which are numerous. These nodules break down to form ulcers.

The hypertensive ulcers or the Martorell’s ulcers are a condition which appear in older aged people and is usually associated with atherosclerosis. This ulcer is extremely painful and is usually punched out and extends down into the deep fascia. It is note worthy that all peripheral foot pulses are present.

The clinical characteristic feature of the Meleney’s ulcer is the burrowing nature, which is the undermining of ulcer with a lot of granulation tissue on the floor. These ulcers are painful and show a tendency to spread and render the patient toxemic.

Ulcers secondary to Paget's disease are mostly situated over the convexity of the anteriorly bowed tibia, with the edges densely adherent to the bone which forms its base. In case of rheumatoid arthritis the ulcer forms due to break down of the nodules.

The size of the ulcer varies, the wound becomes punched out, shallow and is usually painful, without induration and slow to heal.

Ulcers caused by mycotic infections are rare. Primary neoplasms of the skin which may cause ulceration in the lower extremity are squamous cell carcinoma and malignant melanoma.

The occurrence of the squamous cell is most common in the age group of 40 years and above, the risk of contracting it raises as the age increases. In this type of ulceration bleeding is a common complaint. The tumor is painless but becomes painful if it invades deeper structures. The origin of the squamous cell carcinoma is as a tender nodule, which enlarges and the center becomes necrotic and sloughs out. The variation of the size of the ulcer is diverse and the wound is everted and has raised edges. Pale and unhealthy granulation tissue may also be seen. If the ulcer has penetrated too deep then there is a chance that interior structures may also be exposed. The base of the ulcer is indurated, with restricted mobility. Once the tumor is anchored to the underlying structures the regional lymph nodes may become

enlarged. But until it is proved otherwise, it should be assumed that the palpable lymphnodes are due to metastasis.

As explained previously in the malignant melanoma, the patient usually complains of a long-standing mole which has shown rapid growth within a few days. The mole becomes darker and such color change is often patchy. Sometimes malignant melanoma does not show pigmentation and such lesion is called amelanotic ulcer. The overlying epithelium becomes ulcerated and often breaks down. The tumor hence tends to bleed. The tumor cells gradually tend to invade the surrounding skin and thus forming a halo. This condition is not a painful one, but it is often itchy. Only in the later stages does one complain of weight loss, dyspnoea or jaundice. The occurrence of these melanomas is usually in the palms or the soles of the feet. Apart from the bleeding, the surface of the wound looks wet, soft and boggy. The tumor is firm and can be easily lifted from the deeper structures. The regional lymphnodes are often enlarged in malignant melanoma.

In case of hematogenic ulcers, it usually mimics venous ulcers or ischaemic ulcers and are commonly found to occur in anterior part of the leg.

In patients suffering from non-specific yet chronic ulcers, the patients usually have a history of trauma which he might have neglected or for which

he had taken inadequate treatment. The patients in such cases are of poor build, poorly nourished and anemic on examination. These ulcers appear with irregular margins surrounded by oedema and there may be slight tenderness. Often ulcers are foul smelling and with seropurulent discharge. Associated induration, sclerosis and pigmentation are common. Yet another type of ulcers usually occurring in older aged, bed-ridden patients is the Decubitus ulcers. Young people with neurologic disease can also be affected with this type of ulcer. The ulcer localizes over bony prominences, the heel region is one of the characteristic sites. These ulcers are present as deep ulcers and are marked with peripheral undermining which can extend to the bone, causing osteomyelitis.

VII. MANAGEMENT

a. INVESTIGATIONS

There have been many investigations done for analysis and understanding of the leg ulcers. These investigations are numerous and varied. These investigations help us to come to a definite diagnosis of the ulcers and may also guide about the outcome of surgery if any.

The investigations can be broadly classified in to three different classes:

a) Routine investigation.

b) Special investigation.

c) Investigation to know vasculature status of the leg ulceration.

a) Routine investigation

The routine investigations that are helpful are the following tests:

- **Hemoglobin (Hb%)**
- **ESR**
- **TC and DC**
- **Haemogram**

b) Special investigation

- **Fasting blood sugar, PPBS, and GTT**

As we know diabetes mellitus is one of the many contributing factors for the formation of ulcers in legs and for peripheral neuropathy.

- **Bone Marrow Aspiration**

  Is recommended in cases of suspected leukaemia or aplastic anemia .

- **Lipid profile**

The detection of the atherosclerotic condition is possible when this particular test is performed.

- **Blood VDRL test**

The VDRL test is a non-specific flocculation test used for diagnosis of syphilis, Yaws Pinta and Bejel.

- **Liver function test**

  To evaluate Liver Function

- **Culture and sensitivity of Bacteria/fungus**

The pus from the ulcer is sent to the lab for culture and sensitivity for bacteria or fungus as the case may be.

The presence of these pathogens in the pus does not have to mean that they are the causators. These pathogens only act as catalysts and worsen the condition and hinder essential processes such as coagulation and fibrinolytic activity from taking place.

Presence of other bacteria must be taken more seriously. These bacteria are:

a) Heamolytic Steptococci.

b) Fusiform bacilli and spirochete in hot humid conditions.

c) Candida albicans.

d) Pseudomonas aeroginosa.

e) Proteus

f) Staphylococcus

Presence of these organisms needs attention. They have to be treated with proper antibiotics.

- **Biopsy and histopathological examination from ulcer site**

Malignant skin changes are common in chronic leg ulcers. A biopsy should be taken from all suspicious ulcers or ulcers that do not respond to appropriate treatment. Also, chronic ulcers need to be biopsied at regular intervals as malignant change in these ulcers in directly related to their duration (Yang D. Morrison B. D. et al and Smith J,
Mello I. F et al).[16, 17]

- **Radiological examination**

X-ray should be taken to find out any bony abnormalities and or osteomyelitis of the underlying bone.

HANDHOLD VASCULAR DOPPLER

DUPLEX SCAN

c) INVESTIGATION OF VASCULAR STRUCTURE OF THE LEG

- **Investigation of Venous Disease**

Doppler ultrasound

A Doppler flow probe can be used to exclude arterial disease and to determine patency of a vein and a bi-directional probe is used to detect any reflux. This investigation is carried out with patient standing. Doppler probe is first placed on saphenofemoral junction (SFJ) and blood flow is assessed to located venous flow in common femoral veins. With one hand the examiner gently heard as “woos” from loud speaker of the Doppler machine. The calf compression is released and any backflow is noted. The probe may be also held in sapheno-popliteal junction (SPJ) while calf is, compressed and released to test competence of veins in this region.

Duplex ultrasound imaging

This technique involves use of high-resolution B-mode ultrasound imaging and Doppler ultrasound to obtain images of veins and simultaneously measure the flow. It provides both functional and anatomical information. Modern duplex machines represent flow as a colour map which is superimposed on grey scale image of the vessel. This technique is highly reliable in the investigation of arteries and veins, and is most appropriate when detailed analysis of anatomy and physiology of venous system is required. Blocked or incompetent veins can be easily identified. The origin

of varicose veins and venous ulceration can be identified and in patients with deep vein thrombosis the thrombus can be seen (Seurr John. H et al).[22]

Venography

This investigation is X-ray equivalent of Duplex ultrasonography. It has been largely replaced by duplex imaging.

Plethysmography

a) Photo- Plethysmography: in this investigation a probe is attached to the skin to asses venous refilling of surface venules by measuring light transmission of the skin. The filling of vessels reflects pressure in the superficial veins of the leg. The patient sits till the trace stabilizes. Then he performs a series of 10 dorsiflexion at the ankle. The venous pressure falls as venules empty and the trace falls. The patient sits and venous refilling occurs only through arterial inflow, a slow process taking 20 to 30 seconds when the limb is at rest. In venous incompetence filling occurs through venous reflux also which speeds the filling time. This test can be repeated after applying tourniquet at the thigh to occlude long saphenous system and below the knee to occlude short saphenous system to know which set is incompetent.

b) Other forms of Plethysmography like air plethysmography, light reflex rheography and strain gauge plethysmography are used only

by experts in laboratories to quantify venous function caused by incompetent veins. (Seur John. H et al).[18]

- **Investigations For Arterial Disease**

Doppler ultrasound technique

The frequency of the ultrasound waves is altered when it bounces off moving objects and the change in the frequency can be directly related to the velocity with which the object is moving, this effect is termed as the Doppler'seffect.

Duplex imaging (Duplex ultrasound scanning)

This is an investigative technique of major importance in vascular disease. A duplex scanner uses 'B' mode ultrasound to provide an image of vessels. This image is created through the different ability of different tissues to reflect the ultrasound beam. A second type of ultrasound, namely Doppler ultrasound is then used to insonate the imaged vessels and the Doppler shift is analyzed by a computer in the duplex scanner itself. Such shifts can give detailed knowledge of vessel blood flow, turbulence etc.,

It also allows the cross sectional area of arterial lumen to be measured. By use of colour, the flow towards or away from the transducer can be easily distinguished so that peripherally running arterial flow (red) can be immediately distinguished from centrally directed venous flow

(blue); the intensity of colour increases with the velocity of flow. Reversal flow, in venous reflux can be recognizable by a change in colour, so that incompetent veins and leading walls show up as ‘red’ at the moment of reflux.

Duplex scanning is feasible and reliable in detecting crural and pedal artery lesions in lower limb with severe ischaemia (Karacagil. S. Lofberg. A. M).[19.]

Radioisotope tests

Fibrinogen labelled with radioactive isotope I125 is injected into the patient. This is converted into fibrin by thrombin. Fibrin is deposited in thrombus. Various other tests are also performed in this type of special investigations. Some of these are mentioned below:

Arteriography

Arteriography is indicated only when decision has been taken that intervention is needed. Arteriography involves injection of radio opaque solution into the arterial tree using retrograde percutaneous method involving femoral (Seldinger technique). Direct angiography by puncturing the aorta is hazardous and outdated. Hazards include the thrombosis, arterial dissection, haematoma and anaphylaxis (Murie. John).[20]

Digital Subtraction Angiography (DSA)

This technique involves a computer system to digitize angiographic information. It allows image before contrast injection to be subtracted from contrast image, yielding

great clarity. This avoids arterial puncture although high volumes of contrast agents are required.

Plethysmography

This assesses volume change in a limb with each cardiac cycle. Air filled cuffs or mercury in rubber strain gauges have been typically used. For most clinical purposes the test has been superseded by duplex imaging (Murie John).[20]

b. TREATMENT

General Treatment

In treating the ulcer the surgeon must first recognize and treat any systemic condition that inhibits the healing. Systemic diseases, particularly malignant tumors, diabetes mellitus, nutritional deficiencies and some medication can retard the wound healing. In addition deficiencies of vitamin C, zinc, iron and protein can also retard healing as these are the nutrients needed to synthesize collagen. Avoidance of smoking is necessary for wound healing.

The elevation of the affected limb well above the heart level is an excellent treatment method and the limb must be allowed to remain in this state for at least 2 hours. The surgeon must encourage the performance of exercise. The rate of exercise should be around 40/min and this is enough to empty the veins without overloading the lymphatic system. As a rule elevation must not be done in cases of ulcers with arterial origin.

Systemic treatment

Infection of the ulcerous wound greatly hampers the wound healing. Oral agents such as decloxacillin are appropriate treatment for gram positive infections, while Amoxycillin and clavulanate (Augmentin) will extend bacterial coverage to some gram negative organisms and anaerobes but not to pseudomonas, which can be effectively treated with Ciprofloxacin.

Vasoactive medication

Nifedipine is effective in Raynaud's disease as a result of its ability to restore blood flow to the digits. Ketanserine, an S-serotoninergic blocker is helpful in treating ischaemic ulcers. Pentoxifyline (trental) is effective in treatment of the above conditions.

Ketanserine has also been shown to increase the rate of ulcer healing in diabetic ulcers (O'Meara S.O. Cullum N et al).[21]

Local Treatment

Local Application

This method is practiced to soak up exudates, remove slough, control infection, sooth the surrounding skin, promote granulation tissue and protect seeding of new epithelium as well as to relieve pain.

The use of topical antibiotics must be restricted as most of these are strong sensitizers. The use of antiseptic agents is safer and acceptable.

Wet dressing using Eusol or 0.5% Acetic acid, chlorhexidine or 0.5% silver nitrate have been shown to be inhibitory to granulation tissue, and must be advised with caution. However, Eusol is a useful wetting agent for softening and removing superficial slough, especially when there is no

granulation tissue. 0.5% silver nitrate is recommended when the granulation tissue is over exuberant.

Gilliland E. L and Wolfe John H.N.[4] recommended povidone as the antiseptic for general use and silver sulphadizine for short periods of pseudomonas species have been cultured and potassium permanganate (1/8000 dilution) for wet ulcers with surrounding eczema but again only for limited periods as it can cause local hyperkeratosis.

In recent times new topical dressing have gained wide acceptance in the treatment of leg ulcers. The hydrocolloid dressings are to debride the ulcer, while some of the polyethylene dressings have the advantage that they decrease the pain in the ulcers. These dressings do not increase the risk of infection in the ulcer colonized with bacteria.

Carboxy-methyl-cellulose dressings are safe, effective and well tolerated in management of non-ischaemic, non-infected deep diabetic foot ulcers (Piaggesi A., Baccetti F et al).[22]

Compression bandages

The advantage of compression lies in the provision of adequate counter pressure when the patient continues normal occupation and normal life. Several methods of compression have been used. It probably maters less which method is chosen that it is carried out with expertise and that its

limitations are known. Recently the group from Charing Cross Hospital has achieved 80% of ulcer healing within 6 weeks using a high compression 4-layer bandage. Compression is more effective in healing venous leg ulcer. High compression hosiery is more effective than moderate compression in preventing ulcer recurrences (Callam N, Nelson E. A et al).[23]

SURGICAL TREATMENT

Many ulcers that are infected and do not heal require wound toileting debridement and slough excision. Those ulcers which still do not heal by secondary intension or have delayed healing or those which have large areas of defects require skin grafting.

Grafts

Primary indication for surgical treatment is the failure of conservative line of management. It is also helpful in cases of extensive ulcers which when treated conservatively will take long drawn-out period for healing; that too with fibrosis and scarring.

Pinch grafting

It is relatively a painless procedure that consists of removing small “pinches” of skin, partial thickness grafts, under local anesthesia from a donor site, e.g.: the thigh.

SPLIT SKIN GRAFTING

Split thickness grafts

These can be used in larger ulcers. It is advisable to use a graft that allows the underlying build-up of exudates to escape. The meshed graft heals by secondary intension, which results in irregular surface of the grafted site.

Keratinocyte graft

Autografts: Human keratinocytes from a small skin biopsy specimen can be grown in vitro, in 2 to 3 weeks, to form sheets of confluent epithelium.

Allografts: Culture of epithelium derived from an allogenic donor (new born fore-skin) and subsequent application to chronic non-healing ulcer are fairly recent developments.

In chronic wound such as ulcers, however both auto grafts and allografts seem to act as temporary biologically active wound dressings. It is likely that growth factors released by the keratinocytes stimulate healing. Also cultured epidermal allografts hasten healing of refractory leg ulcers apparently by stimulating the migration and / or proliferation of acceptors keratinocytes rather than through take up of the graft (Beele H., Naeyaert J. M. et al).[25]

SPECIFIC TREATMENT

Venous ulcers

Specific treatment of venous stasis diseases must first be directed towards reducing venous hypertension. The hypertension can also be reduced through the use of intermittent pneumatic compression.

For stasis ulcers that are refractory to these treatments, some of the surgical approaches include excision of the incompetent valves, leg valves reconstruction or transplantation of the brachial vein valve to leg veins.

Patients who were suffering from varicose ulcers were first treated for ulcer with limb elevation, compression bandaging (Bisgaard's line of treatment). Later the patients were posted for surgery - Trendelenburg's operation (i.e. flush ligation of S.F. junction) and stripping (Meyers stripping); if there is saphenofemoral incompetence or multiple ligation and excision of vein, if perforators are incompetent.

Arterial ulcers

The ulcers result from the localized occlusion of small arterial branches, then some will heal solely with bed rest with elevation of the head end of the bed and cool saline compression.

Others will respond to lumbar sympathectomy combined with complete excision of the ulcers and its surrounding rim of infracted tissue

BELOW KNEE AMPUTATION

followed by a split thickness skin graft. Occasionally if arterial bypass surgery may be warranted.

Transluminal angioplasty may be performed for occlusive disease by inserting a balloon catheter into an artery and inflating it within a narrowed area. Artherectomy can be performed by a variety of new devices available to allow the percutaneous removal of atheroma from within the vessel.

Diabetic ulcers

Proper assessment of the diabetic status, severity of infection and general nourishment is the key to the success of management of ulcers. Regardless of the type of diabetes mellitus, they are suffering from (IDDM or NIDDM) all patients must be put on soluble insulin therapy. Depending upon the blood and urine sugar measurement doses should be adjusted.

Malignant ulcers

Management of squamous cell carcinoma is primarily by Wide excision (2cm clearance in depth and width), which is the treatment of choice once the diagnosis is confirmed by biopsy. Superficial radiotherapy may cure 80% of early lesions. .Treatment of malignant melanoma is primarily surgical, excision with 1-2cm clearence.

When the lymph nodes are significant clinically as well as pathologically radical block dissection is justified

MATERIALS AND METHODS

MATERIALS AND METHODS

The material for this study was drawn patients admitted to the Surgical Department, Govt. Royapettah Hospital, Kilpauk Medical College, Chennai. During the study period between June 2007 to Nov. 2009.

A total number of 200 cases of leg and foot ulcers were considered for this study. This group was a diversified one and included patients of both sexes and of all ages, all religion and economic strata. This study included cases of stasis ulcers, diabetics with leg ulcers, traumatic ulcers, arterial ulcers and others.

A detailed history was collected with particular reference to onset, duration and type of lesion, socioeconomic strata and occupational factors and systemic diseases. Any histories of similar ulcers were also noted.

A thorough systemic and local examination was carried out. The morphological features of ulcers i.e. - number, distribution of ulcer on leg or foot site and associated diseases like varicose veins, eczema or patches were noted.

But while presenting only relevant positive and some important negative findings were shown to make the study brief and to avoid unnecessary repetitions.

ANALYSIS AND OBSERVATIONS

ANALYSIS AND OBSERVATIONS

Table 1

Distribution of various types of chronic leg and foot Ulcers

Total No. of patients studied: 200

Sl. No	Etiological Type	No. of patients	Percentage
1.	Diabetic ulcer	68	34%
2.	Venous ulcer	48	24%
3.	Traumatic ulcer	32	16%
4.	Arterial ulcer	24	12%
5.	Malignant ulcer	10	5%
6.	Trophic ulcer	6	3%
7.	Other ulcers	12	6%

Among the 200 cases studied the commonest was found to be diabetic ulcer accounting for 68 cases (34%) followed by venous ulcer (24%), traumatic ulcer (16%), arterial ulcer (12%) malignant ulcer (5%), tropic ulcer (3%) and others 12 (6%).

According to Gilliland 95% of leg ulcers are due to vascular etiology and venous ulcers dominates accounting for up 90% of the cases. Arterial ulcers account for 5 & 10% and others are due to neuropathy or a combination of both (Young RJ).[10]

Incidences of various chronic ulcers are shown graphically in graph No.1 above.

GRAPH 1

SHOWING DISTRIBUTION OF VARIOUS TYPES OF LEG AND FOOT ULCERS

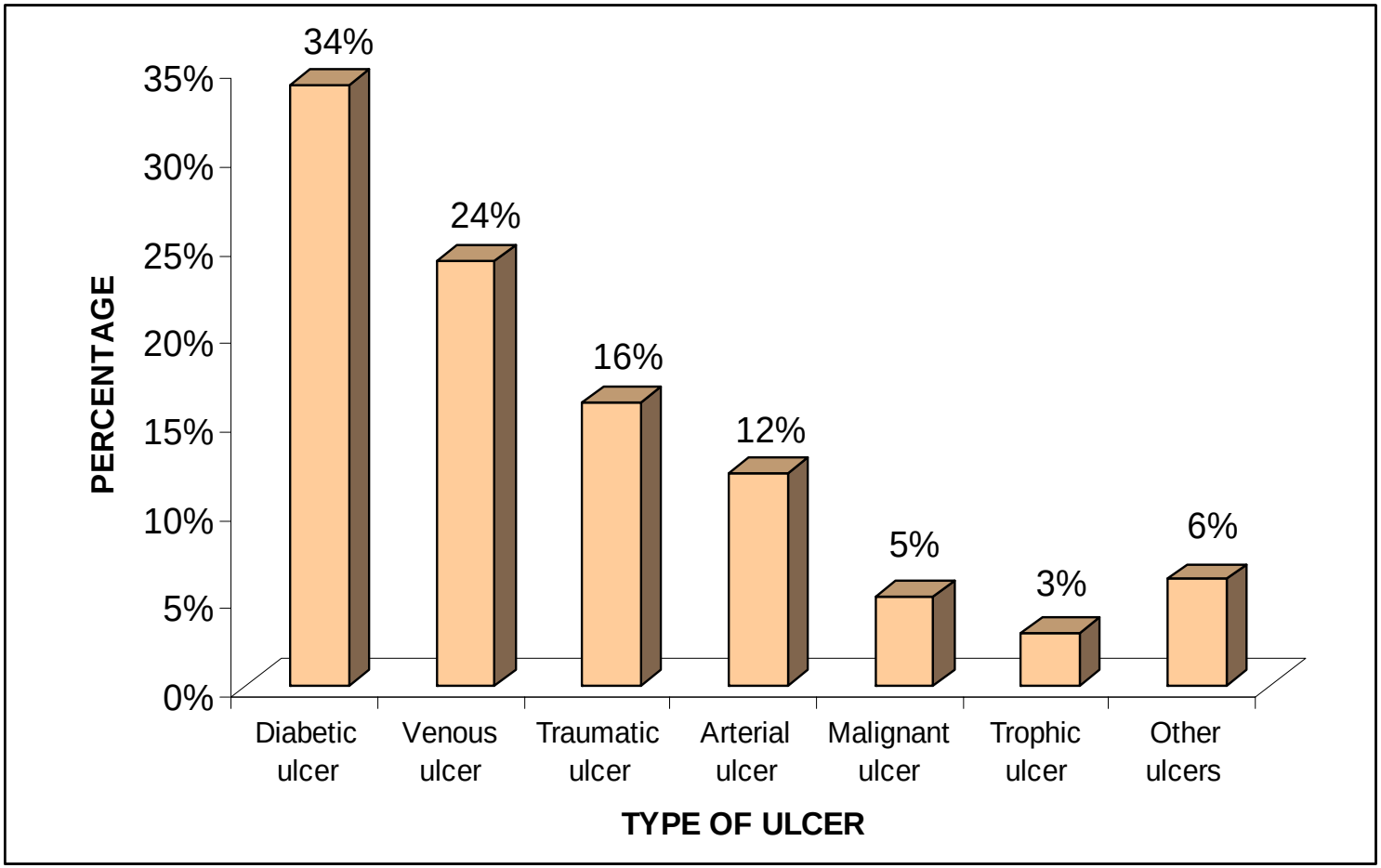


Table 2

Sex distribution of various types of chronic leg and foot ulcers

Sex	No. of cases	Percentage
Male	**172**	**86%**
Female	**28**	**14%**

The above figures indicate that chronic leg ulcers were more common in males than in females – males accounting for 86%. However, in other studies there has been no gross difference between male/female ratio.

Table 3

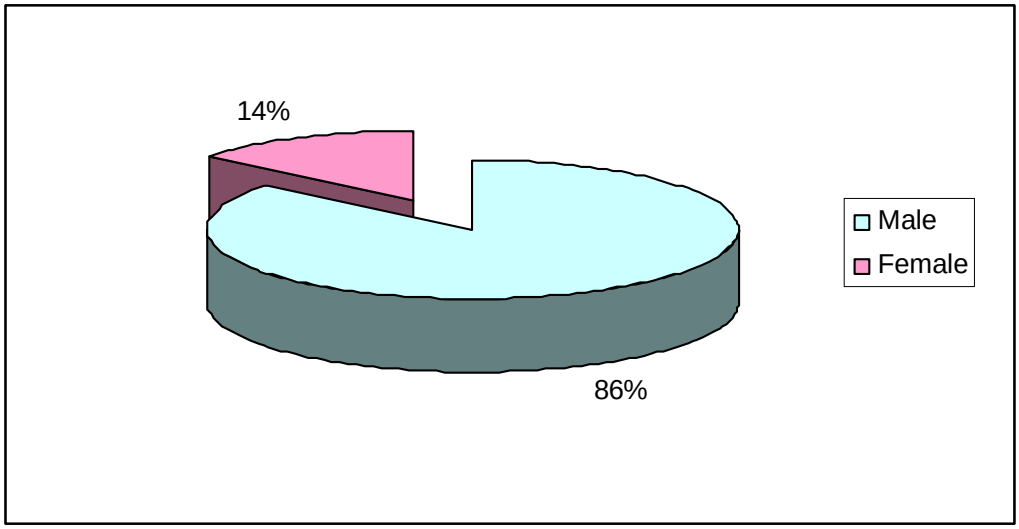
Age distribution of various types of chronic leg and foot ulcers

Sl. No	Age group	No. of cases	Percentage
1.	Up to 10 Years	0	0%
2.	11-20	2	1%
3.	21-30	11	5.5%
4.	31-40	48	24%
5.	41-50	48	24%
6.	51-Above	91	45.5%

Incidences of leg ulcers in this study group were found to be maximum in the age group of 51 & above.

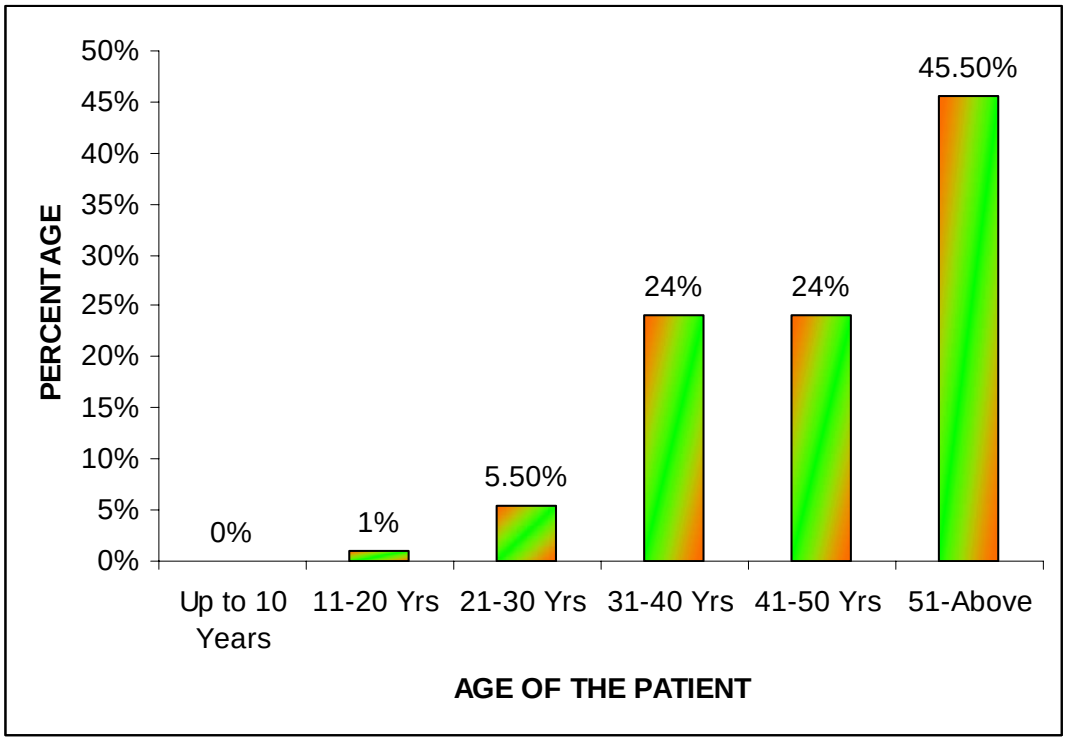
GRAPH 2

SHOWING SEX DISTRIBUTION OF ULCERS



GRAPH 3

AGE DISTRIBUTION OF VARIOUS TYPES OF CHRONIC LEG AND FOOT ULCERS



The youngest patient was 19 years old and the oldest were 80 years old. Cornwall et al[27] in their study had 70% of the patients over the age of 70 years and according to a study done by Callam MJ[23] ulceration began before the age of 40 years in 22% of the patients.

Ulcers Associated with Diabetes Mellitus

Out of 200 cases studied ulcers associated with diabetes mellitus accounted for 68 cases.

Table 4
Distribution of diabetic ulcers in the limbs

Sl. No	Affected Limb	No. of cases	Percentage
1.	Right limb	32	47.6%
2.	Left limb	34	50%
3.	Bilateral	2	2.94%

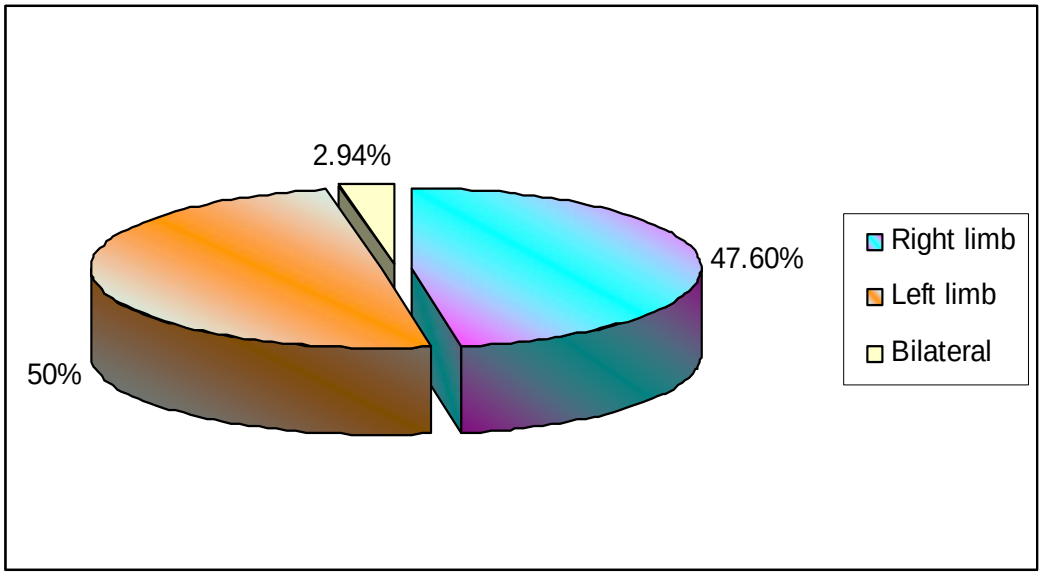
From the above study, it is noted that diabetic ulcers were relatively common in the left limb accounting for 50% of cases.

Table 5

Sex distribution of diabetic ulcers

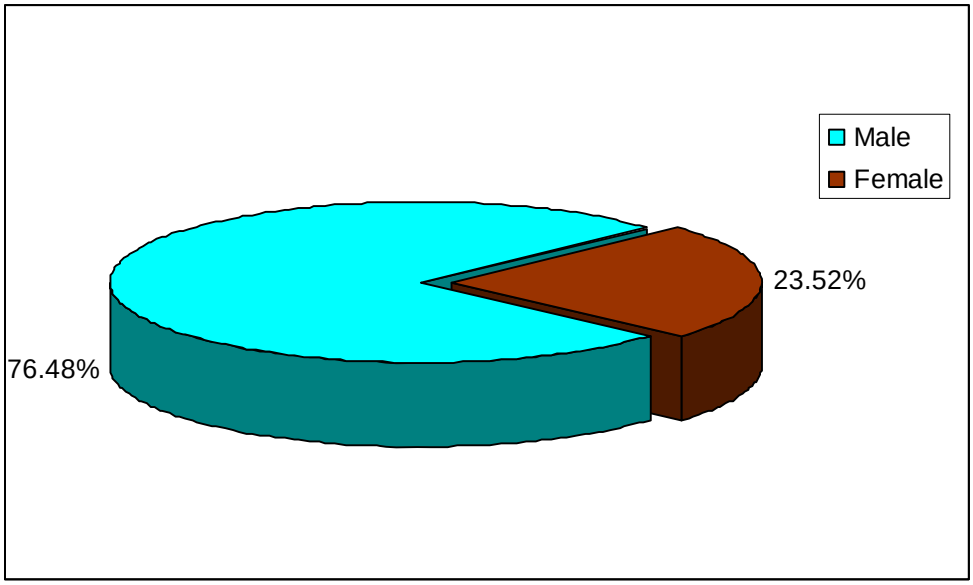
Sex	No. of cases	Percentage
Male	52	76.48%
Female	16	23.52%

GRAPH 4
DISTRIBUTION OF DIABETIC ULCERS IN THE LIMBS



GRAPH 5

SHOWING SEX DISTRIBUTION OF DIABETIC ULCERS



From the above study, it is noted that diabetic ulcers were relatively common in males accounting for 76.48% and less common in females accounting for only 23.52%.

Table 6

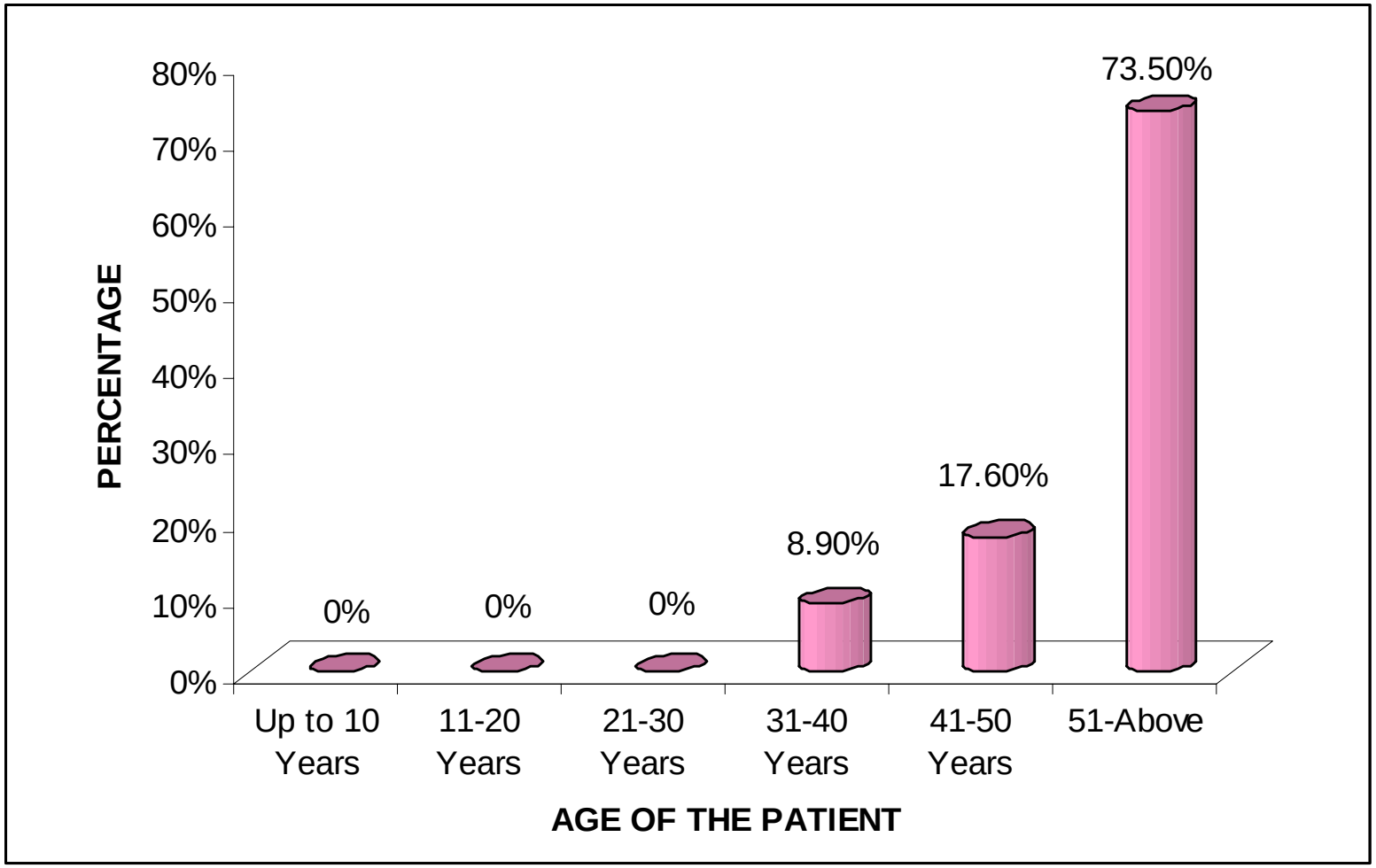
Age distribution of diabetic ulcers

Sl. No	Age group	No. of cases	Percentage
1.	Up to 10 Years	0	0%
2.	11-20 Years	0	0%
3.	21-30 Years	0	0%
4.	31-40 Years	6	8.9%
5.	41-50 Years	12	17.6%
6.	51-Above	50	73.5%

As noted above the maximum no of patients suffering from diabetic ulcers were in the age group of above 50 years accounting for about 73.5% of the cases.

GRAPH 6

AGE DISTRIBUTION OF DIABETIC ULCERS



Venous Ulcers

Out of the 200 cases studied ulcers associated with venous causes accounted of 48 cases.

Table 7

System affected in venous leg ulcers

System	No. of cases	Percentage
Long saphenous	31	64.5%
Short saphenous	3	6.2%
Both	10	21.0%
Deep veins	4	8.3%

In this study, long saphenous system was found to be by far the commonest system affected in case of venous ulcers accounting for 64.5%.

Table 8

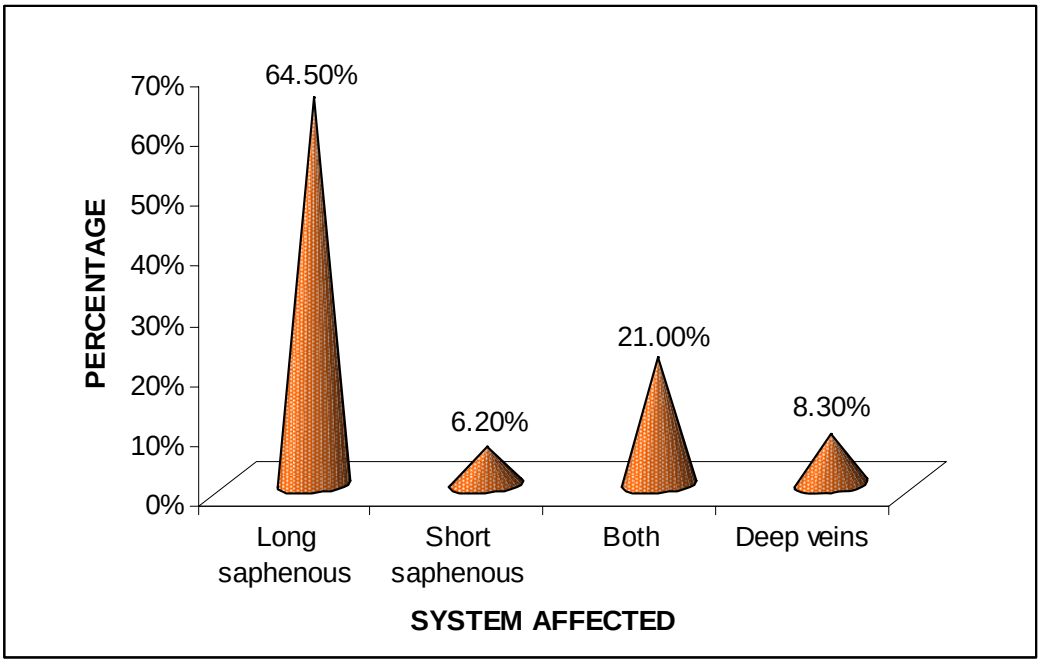
Age distribution of Venous Ulcers

Sl. No	Age group	No. of cases	Percentege
1.	Up to 10 Years	0	0%
2.	11-20 Years	0	0%
3.	21-30 Years	6	12.6%
4.	31-40 Years	23	47.9%
5.	41-50 Years	11	22.9%
6.	51-Above	8	16.6%

Venous ulcers were found to be the commonest between the age group 31 – 50 years.

GRAPH 7

SHOWING DISTRIBUTION OF VENOUS ULCERS WITH REFERENCE TO SYSTEM AFFECTED



GRAPH 8

AGE DISTRIBUTION OF VENOUS ULCERS

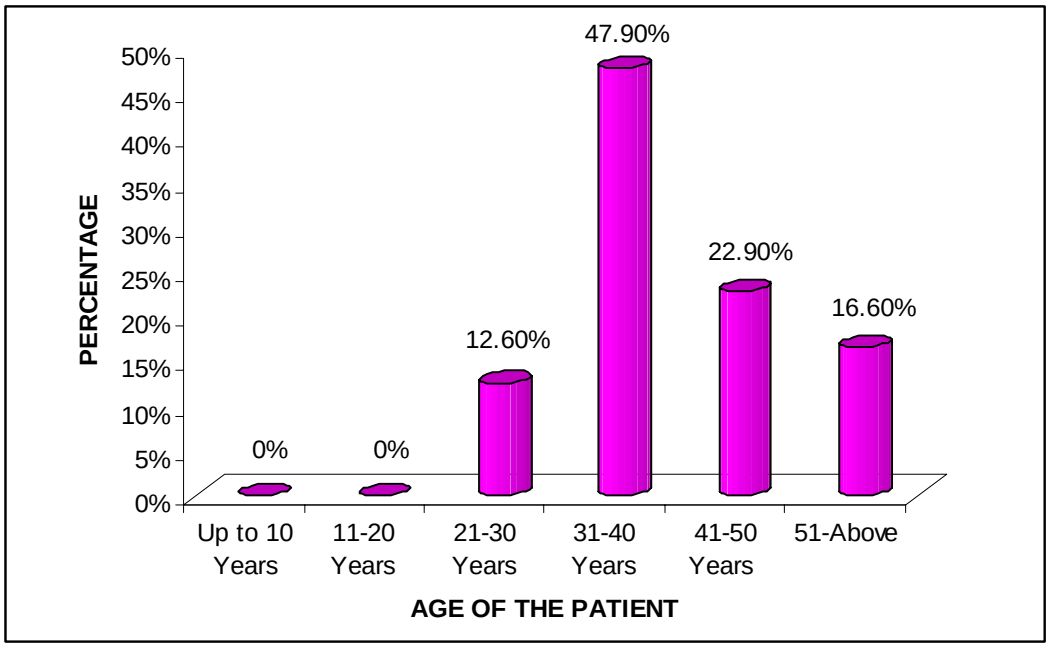


Table 9

Sex distribution in Venous Ulcers

Sex	No. of cases	Percentage
Male	44	91.67%
Female	4	8.33%

Males were more commonly affected accounting for 91.67%. In other published studies it is noted that females have a slightly more preponderance over males.

Arterial Ulcers

Out of 200 cases, 24 were arterial ulcers.

Table 10

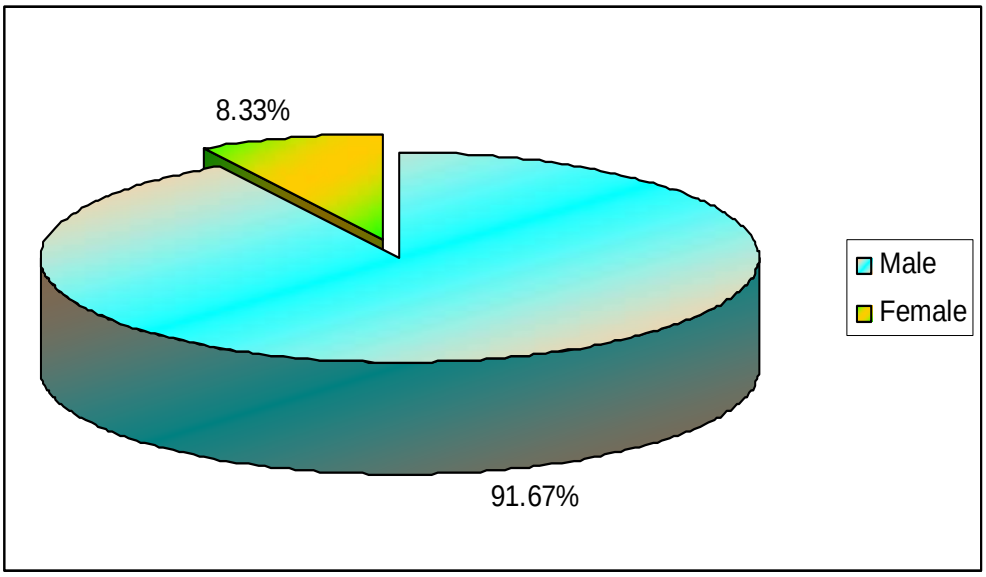
Age distribution of various types of arterial ulcers

Sl. No	Age group	No. of cases	Percentage
1.	Up to 10 Years	0	0%
2.	11-20 Years	0	0%
3.	21-30 Years	0	0%
4.	31-40 Years	5	20.83%
5.	41-50 Years	11	45.83%
6.	51-60 Years	5	20.83%
7.	61-70 Years	3	12.5%

Arterial ulcers were found to be the most common ulcers in the age group of 41 to 50 years.

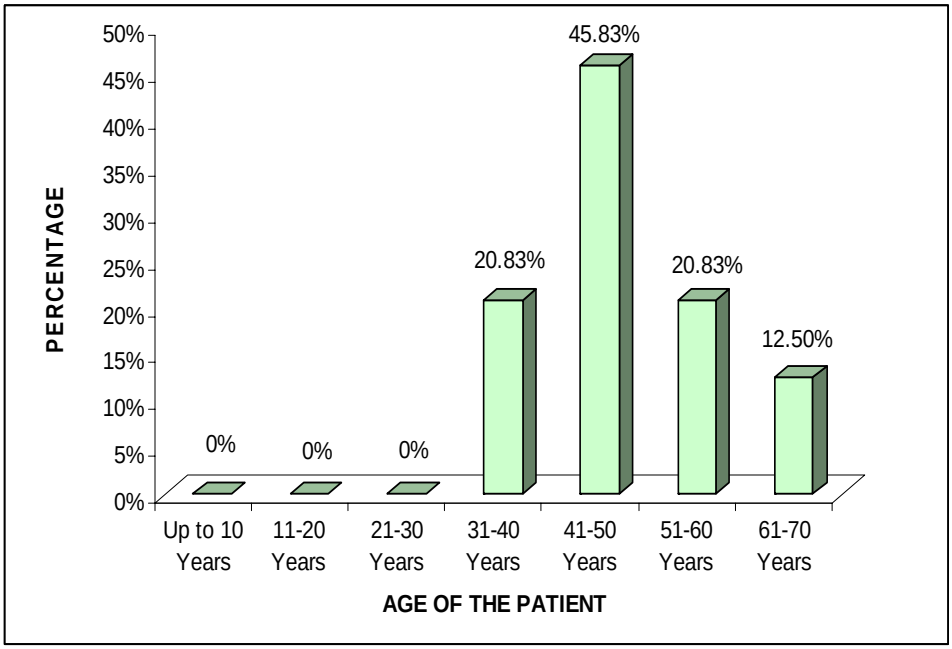
GRAPH 9

SEX DISTRIBUTION IN VENOUS ULCERS



GRAPH 10

AGE DISTRIBUTION OF VARIOUS TYPES OF ARTERIAL ULCERS



Peripheral vascular diseases are 7 times more frequent in 60-year-old when compared to 70years olds according to Hanson Carita.

Table 11

Pathology in arterial ulcers

Pathology	No. of cases	Percentage
TAO	10	41.6%
Atherosclerosis	14	58.4%

Atherosclerosis was found to be the commoner association with arterial ulcers constituting 58.4%. The only other association with arterial ulcers was TAO accounting for 41.6%.

Traumatic Ulcers

A total no. of 32 traumatic ulcers were noted in the study group out of which 6 were associated with anemia. One of these ulcers was present on the joint surface. One of these ulcers was result of secondary infection following primary closure by suturing. The rest of the ulcers were that of avulsive type with some degree of skin loss.

GRAPH 11

PATHOLOGY IN ARTERIAL ULCERS

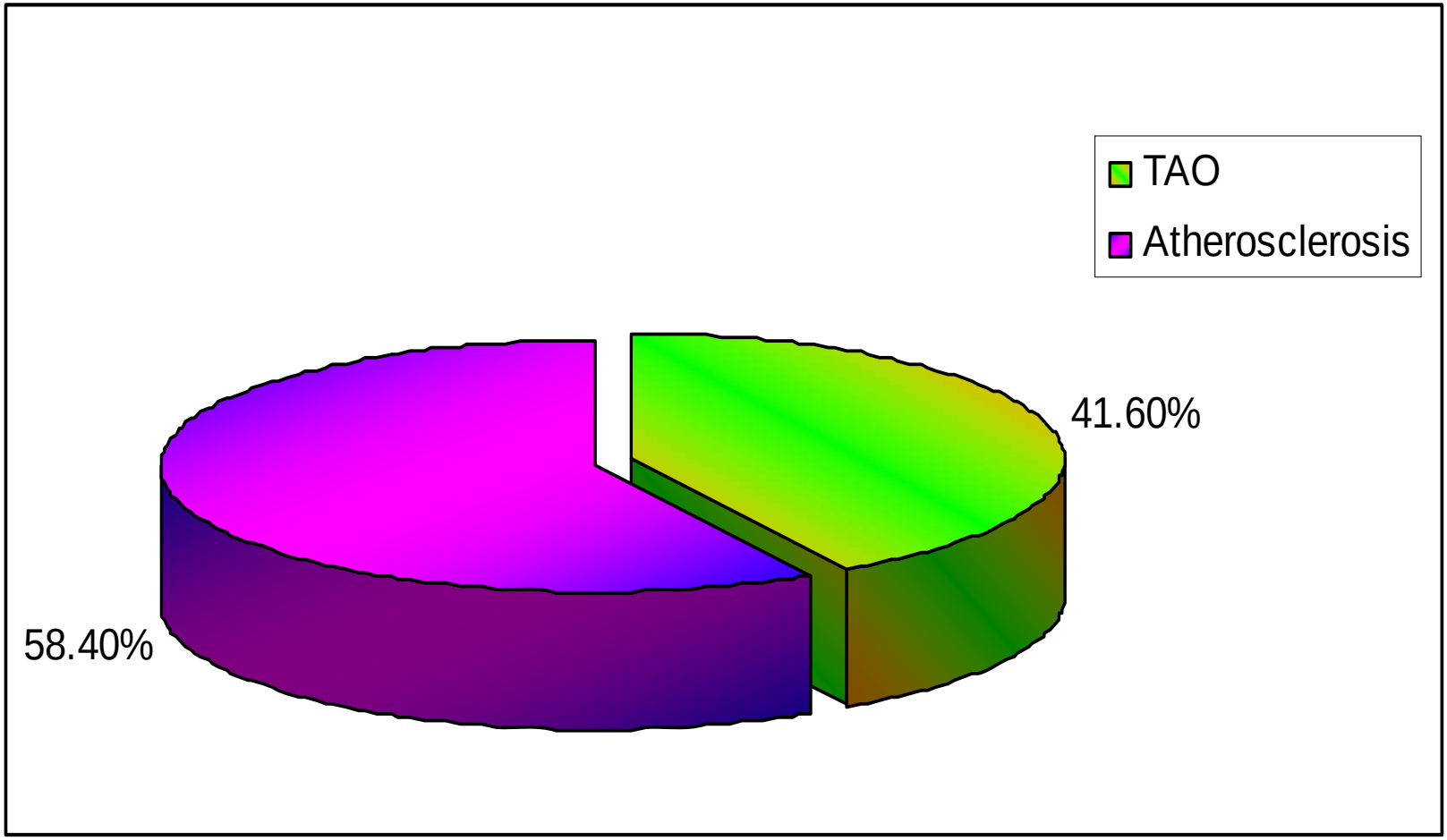


Table 12

Location of the ulcer according to its types

Sl. No	Type of ulcer	Gaiter zone	Foot	Leg	Total
1.	Diabetic	0 (0%)	60(88.3%)	8 (11.7%)	68
2.	Venous	42(87.5%)	1 (2.1%)	5 (10.4%)	48
3.	Arterial	0 (0%)	24 (100%)	0 (0%)	24
4.	Malignant	0 (0%)	6 (60%)	4 (40%)	10
5.	Others	1 (8.33%)	8(66.66%)	3 (25%)	12

The venous ulcers occurred more commonly in the gaiter zone (87.5%). Where as arterial and diabetic ulcers occurred mainly in the foot i.e., 100% and 88.37% respectively. About 60% of malignant ulcers occurred in the foot and rest of 40% in the leg.

According to Hanson Carita ulcers below the line of shoe and feet are considered mostly caused by arterial insufficiency and or diabetes. Ulcers on the gaiter zone are mostly caused by venous insufficiency.

Table 13

Types of bacteria isolated from the ulcers

Sl. No	Pathogen	No. of cases	Percentage
1.	Staphylococcus	26	28.9%
2.	Klebsiella	18	20%
3.	Proteus	14	15.5%
4.	Streptococcus	20	22.2%
5.	Pseudomonas	3	3.3%
6.	No growth	4	10%

Only 90 cases were sent for culture and sensitivity tests. Staphylococcus was found to be the most common pathogen accounting for 28.9% of the bacteriological isolates. This was followed by proteus, which accounted for 15.5%, Klebsiella which accounted for 20%, streptococcus and pseudomonas accounting for 22.5 % & 3.3% each.

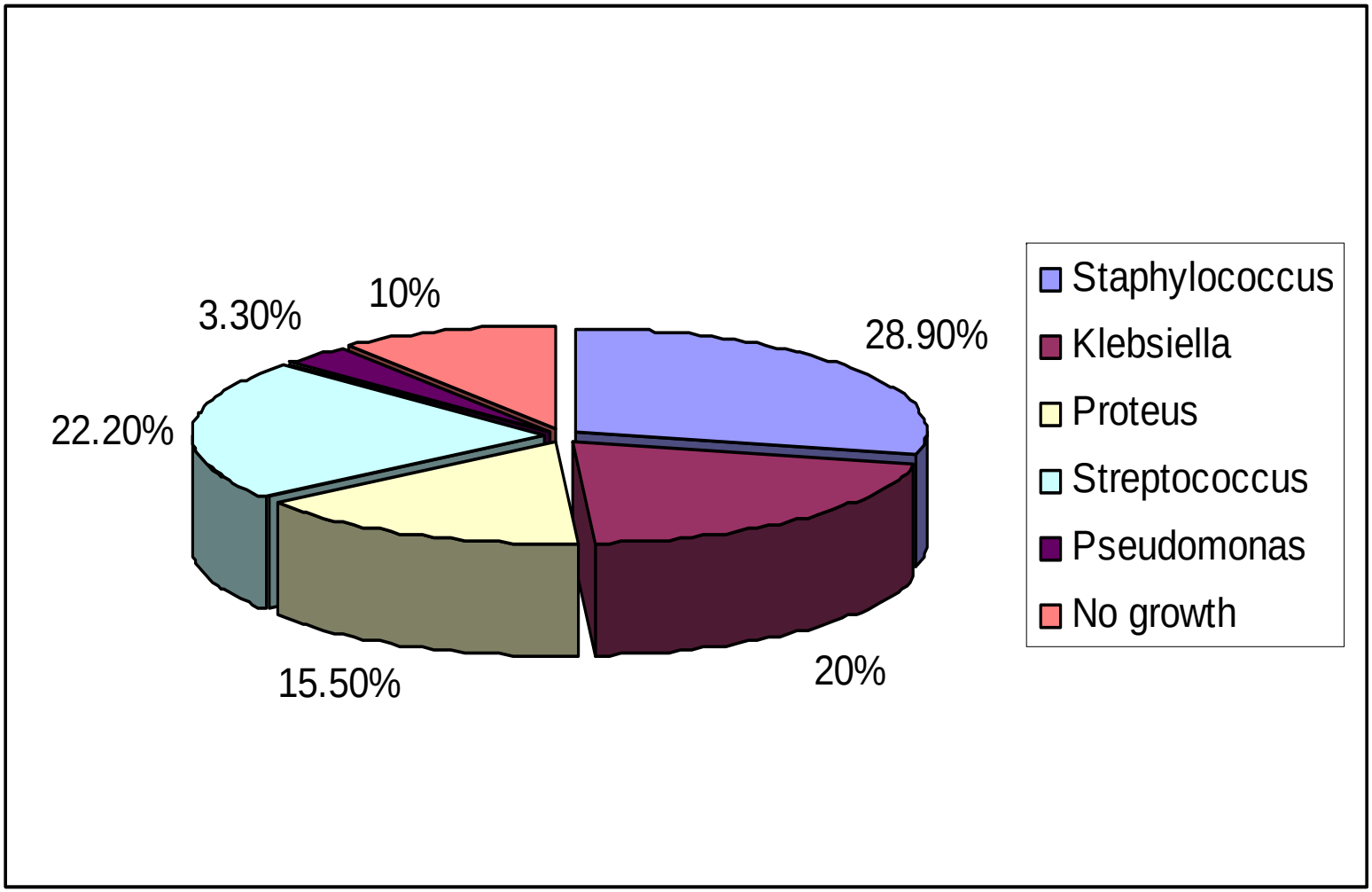
Staphylococcal infection is the most common infection is diabetic foot.

Most foot infections are polymicrobial, staphylococcus is recovered from 33 to 50% of the cases (Norman Weinszweig and Raymond M. Dunn).

Most of the patients in this study group belong to the lower socio-economic status.

GRAPH 12

SHOWING TYPES OF BACTERIA ISOLATED FROM THE ULCE



DISCUSSION

DISCUSSION

The prevalence of leg ulcers is probably between 0.18% and 1% (Phillips, Tania et al).[5] 95% of leg ulcers are due to vascular etiology, (Gilliland)[7] and among all chronic wounds lower extremity venous ulcer dominates the differential diagnosis accounting for up to 90% of the cases (Burton S. Claude)[8] (Callum M. J. et al).[9] Arterial diseases account for 5% to 10%, most others are due to neuropathy or a combination of both (Yound J. R).[10]

In this study chronic ulcer with vascular etiology accounted for only 36% of all chronic ulcers. Out of this venous ulcers accounted for 24% and arterial ulcers accounted for 12%. Chronic ulcers associated with diabetes accounted for nearly 34%. Traumatic ulcers accounted for 16% of the cases. Malignant ulcers accounted for 5% and other ulcers for 6%.

As observed above the present study was not comparable with the published studies mentioned probably because of following reasons:-

- The study group of 200 patients was too small a number to draw any comparative conclusions.
- The other published studies were population based, controlled randomized or a group-based study which included different specialties where as this study was a nonrandomized and uncontrolled study.

Some investigators have classified diabetic ulcers as metabolic. The most important factors responsible for causation of ulcer in diabetes are the arterio-sclerotic lesions in large leg arteries and or neuropathy resulting in decreased sensation. If diabetic ulcers in our study are considered vascular disorders rather than metabolic, the percentage of vascular ulcers in our study is about 66% - somewhat comparable to the above study. However, this is controversial and in diabetes it is a combination of factors that are to be considered in causation of leg ulcers.

Also according to Yound J. R.[7] and Boyd A. M. et al,[26] the distribution of different type of ulcers in different studies varies – 70% to 90% for venous ulcer, 5% to 15% for arterial ulcers and 1% to 5% for other ulcers.

As per studies done by Hansson Carita [8] on leg and foot ulcers, ulcers below the line of shoe and feet are considered mostly to be caused by arterial insufficiency and or diabetes. Ulcers on the medial aspect of the ankle in the gaiter zone are mostly caused by venous insufficiency.

In the present study, ulcers had the same site of distribution i.e., ulcers in the gaiter zone were mostly caused by venous insufficiency and ulcers in the foot below the line of shoes were mostly caused by arterial insufficiency and or diabetes.

About 42% of patients in our study had ulcers in the foot only. This is rather high figure in comparison to Hansson's study which showed about only 30% of the ulcers in the foot. This is probably due to more number of diabetic and arterial ulcers in our study.

Cornwall et al[31] in his study had 70% of patients over the age of 70 years. The median age of all patients in this study was 45 years and 44% of the patients over the age of 45 years and had 70% of the patient over the age of 70 years. But according to study done by Callam M. J.[6] the elderly are not the only population at risk: In his study ulceration began before the age of 40 years in 22% of the population studied. In our study, ulceration began before the age of 40 years in 47.9% of the patients.

Peripheral vascular diseases increase with age and are 7 times more frequent in 60 years old patients when compared to 20 years old. (Hansson Carita). In this study, arterial and venous diseases were found to be maximum in the age group of 31 to 50 years. This discrepancy may be due to the fact that, our study group patients in the above age group belong to the working class and the ulcers they suffer from hamper their working capacity making them seek medical help early. And also venous ulcers were found to be most common in the age group of 31 to 50 years which is rather early

when compared to western studies as most of our patients belong to the working class which involved long hours of standing.

Arterial were found to be more common in the age group of 31 to 50 years which again is rather too early as compared to western studies, since we have in our study a significantly high number of TAO cases which are common in young adults.

In our study, there were more men 86% than women 14% with leg and foot ulcers. However, no differences between sexes were found when age specific relative frequencies for all ulcers were compared.

Elastic crepe bandages are the most important forms of treatment for venous ulcer patients (Rightor M. Myers M. B).[28] In our study all the 48 patients who had venous ulcers wore for elastic crepe bandages stretched to 50% providing of around 14 mmHg compression pressure under one layer. These patients were also subjected to local dressings and Bisgaard's line of management. Once the ulcers healed they were taken up for surgery. Out of the 48 patients, 44 were due to varicose veins and 4 due to deep vein thrombosis. Out of 48 patients with varicose veins, 44 underwent surgery in form of ligation and or Trendelenburg's operation and sub fascial ligation. 4 patient with deep vein thrombosis underwent skin grafting. The mean time

for ulcer healing was 17.2 days. The patient who underwent skin graft had his ulcer healed in 7 days only.

A study of recurrences of venous ulcers could not be made due to inadequate time follow up.

Appropriate anti-diabetic therapy informs of plain insulin (Bovine), antibiotics, the debridement and regular dressings were the important methods of treatment for diabetic ulcers in our study. Out of the 68 patients, 60 patients were managed with regular dressings; antibiotics slough excision and or debridement along with anti-diabetic therapy. Three patients underwent amputation as a life saving measure and one patient expired due to Medical causes. 6 patient underwent skin grafting and had his ulcer healed in 10 days. However, the mean healing time was 26.43 days in overall diabetic ulcers.

CONCLUSION

CONCLUSION

- The highest age incidence of leg and foot ulcers in this study was in the age group of 51years and above (45.5%).
- The median age was 45 years and the mean age was 44.28 years.
- There was a marked male predominance of 86%
- Foot was the most commonly affected region 88%
- 87.5% of venous ulcers were situated in the gaiter zone.
- 88% of diabetic ulcers were situated in the foot.
- 100% of the arterial ulcers were situated in the foot.
- Of malignant and other ulcers 60% were situated in the foot and 40% in the leg.
- Staphylococcus was found to be the most common pathogen to be isolated from the ulcers i.e., 28.9%
- 11 patients with leg ulceration had infective gangrene of deeper tissues and they underwent amputation as a life saving procedure and 2 patients with malignant leg ulceration also underwent amputation.
- Most patients with varicose veins underwent some form of operation i.e., ligation and stripping and or Trendelenburg's operation and sub-fascial ligation following healing of ulcers. No recurrences of ulcers were noted.
- 9 out of 32 patients with traumatic ulcers underwent skin grafting. The mean time for the ulcer to heal in patients who were grafted was noted to be 10.25 days as against 17.6 days of those who did not undergo skin grafting

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Sarkar PK, Ballantynes. Management of Leg Ulcer, Postgrad Med J 2001 672-82.
2. Phillips Tania et al. A Study of the Impact of Leg Ulcers on Quality of Life - Financial, Social and Psychological Implications, J Am Acad Dermatol, 1994; 31: 49-53.
3. Baker SR. et al, Epidemiology of Chronic Venous Ulcers, Br J Surg, 1991; 78: 864-867.
4. Gilliland E. L., John H. N. et al. ABC of vascular diseases: Leg ulcers. Br Med J 303: 776-779.
5. Burton CS, Treatment of Leg Ulcers, Dermatol Clin, 1993; 11: 315-23
6. Callam MJ, et al, Chronic Ulcers of the Leg: Clinical History, Br Med J, 1987; 294: 1389-91.
7. Yound JR, 1983, Differential Diagnosis of Leg Ulcers, Card Vaso Clin, 13: 171-93.
8. Hanson Carita, Studies on Leg and Foot Ulcers, Stockholm, Acta Derm Venereol, 1988; 45.
9. Setha KK, SG Darke, Long Saphenous Incompetence as a Cause of Venous Ulceration. Br J Surg, 1984; 76: 745-755.
10. Hoare M. C. et al, Role of Primary Varicosities, Surgery, 82:450.

11. Junger M, Steins A, Hans M., Hafner H. M. Microcirculatory dysfunction in Chronic Venous Insufficiency, Dept of Dermatology, University Hospital, Tubigen, Germany-2000.
12. Stacy MC, Burnand KG, Bhogal BS, Black. M., Dept of Surgery, St. Thomas Hospital, London, UK, in Cardiovasc Surg, 2000; Aug-372-80
13. Tibbs J David, Venous Disorders, Vascular Malformations and Chronic Ulcerations in the Lower Limb, Chap 8 in Oxford Textbook of Surgery, Peter J Morris, Ronold A Malt, New York: Oxford University Press Inc., 1994; 505-596.
14. Coffman JD – Intermittent claudication and Rest pain; Physiological concept and therapeutic approaches, Prog-Cardiovsc. P 22-53.
15. Lawrence, B Colen, Mastery of Plastic and Reconstructive Surgery – 1 Editions by Mimis. Cohen, Little brown and Comp, 1994.
16. Smith J, Mello IF et al, Malignancy in Chronic Ulcers and Scars of the Leg (Marjolin's ulcer) – Study of 21 patients in Skeletal Radiol, 2001 June; 331-7
17. Yan D, Morrison BD et al, Malignancy in Chronic Ulcers, Med J Aust, 1996 Jun; 718-20.
18. Scurr John H: Venous Disorders, Chapter 12 in Bailey and Love's Short Practices of Surgery. Charles V Mann, RCG Russell, Norman S Williams, London: Chapman and Hall, 1995; 179-186.

19. Karacagil S, Lotberg A M, Granbo A. et al. Value of Duplex Imaging in Evaluation of Crural and Foot Arteries in Limbs with Sever Ischaemia – Prospective Comparison With Angiography. Eur J Endovasc Surg, Oct 1996: 300-3.

20. Murie, John Arterial disorders Chap 11 in Bailey & love's short practice of surgery. Charles. V.Mann, R.C.G Russels et al: London: Chapmann & Hall 1995; 149-178.

21. O'Meara SO, Cullum. N et al. Systematic Reviews of Wound Care Management – Health Technol Asses, 2000; 4 (21): 1-237.

22. Piaggessi A, Baccethi F et al – Sodium coarboxymethyi cellulose Dressing in Management deep Ulceration of Diabetic foot, Diabet Med, 2001; 320-4.

23. Cullum N, Nelson E et al, Health Technol Asses 2000, 5(9), I – 23.

24. Henry C Vasconex, Mastery of Plastic and Reconstructive Surgery – I Edition by Mimis. Cohen, Little Brown and Comp., 1994.

25. Beele H, Naeyaert, J.M., Goteyn. M et al, University Hospital of Gent, Belgium – Dematologica, 199; 183: 31-35.

26. Boyd AM et al, The Logical Management of Chronic Ulcers of the Leg, Angiology, 1952; 3: 207-215

27. Cornwill J. V., Dore C. J. et al. Leg ulcers – Epidemiology and Etiology, Br J Surg, 1986; 73: 693-696.

28. Rightor M, Myers MB, Cherry G, Relationship Between Oedema and Healing Rate of Stasis Ulcers of the Leg, Am J Surg, 124: 666-668.

PROFORMA

PROFORMA

Case No: IP No:

Name: DOA:

Age: Sex: DOD:

Address: Occupation:

PRESENT HISTORY

Onset: Cough:

Duration: Heamoptysis:

Site: Evening rise of temp:

Number: Difficulty in vision:

Size: Walking bare foot:

Bleeding: Standing long hours:

Examination of Ulcers

Inspection:

Site: Number:

Size: Shape:

Surrounding area: Palpation:

Temperature: Tenderness:

Floor: Edge:

Discharge: Colour:

Bleeding: Base:

Mobility: Depth:

EXAMINATION OF REGIONAL LYMPH NODES:

VASCULAR EXAMINATION:

EXAMINATION OF FOR NERVE LESIONS:

SYSTEMATIC EXAMINATION:

Cardiovascular system:

Respiratory system:

Abdominal examination:

INVESTIGATIONS:

Urine Albumin: Sugar:

Micro:

Blood Hb% TC: DC: ESR:

Blood Sugar: Urea: Creatinine:

Cholesterol: VDRL:

Culture and Sensitivity:

Biopsy:

X- ray chest:

Doppler study:

MANAGEMENT

Conservative

- Antibiotics
- Anti-inflammatory analgesics
- Vasodilators
- Elevation of affected limb
- Active and passive exercise of lower limb
- Alternate day dressing
- Anti-diabetic therapy

Surgical

- Surgical debridement
- Skin grafting
- Subfascialligation
- Ligation and stripping of varicose veins
- Wide excision
- Lumbar sympathectomy
- Amputation

MASTER CHART

KEY TO MASTER CHART

A	→	Antibiotic
AC	→	Anticoagulant
AD	→	Anti-diabetic
Ai	→	Anti-inflammatory
Amk	→	Amikacin
C	→	Compression bandage
Cef	→	Cefotaxime
Cip	→	Ciprofloxacin
Clox	→	Cloxacillin
Coag	→	Coagulase
CP	→	Crystalline Penicillin
D	→	Dressing
DP	→	Dorsalis Pedis
DVT	→	Deep Vein Thrombosis
E	→	Elevation
Ex	→	Exercises
FBS	→	Fasting Blood Sugar
Fem	→	Femoral
GM	→	Gentamycin
Hep	→	Heparin

Inc. Perf	→	Incompetent perforator
Ind	→	Induration
Ing	→	Inguinal
Kleb	→	Klebsiella
L.Sap	→	Long Saphenous
Lig	→	Ligation
LN	→	Lymph node
Mob	→	Mobile
P	→	Purulent
POP	→	Popliteal
Prot	→	Proteus
Pseud	→	Pseudomonas
PT	→	Posterior Tibial
S	→	Serous
S.Sap	→	Short Saphenous
SS	→	Sero-sanguinous
Staph	→	Staphylococcus
Strep	→	Streptococcus
Strip	→	Stripping
Symp	→	Sympathectomy
TAO	→	Thromboangiitis Obliterans